

# Artificial Ventilation

A Basic Clinical Guide

David J. Baker

*Second Edition*



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# Foreword

Artificial ventilation has become one of the essential therapeutic components of resuscitation, making it possible to compensate for one of the main and more frequent organ failures encountered in critically ill patients. Artificial ventilation is also often necessary as a supportive therapy for other conditions, for example circulatory or neurological failure. It has thus become, since the beginning of resuscitation in the 1950s, an essential part of critical care which has developed considerably as a result of very active fundamental, clinical and technological research. These considerable advances have been employed by all doctors who use this technique outside the intensive care unit (ICU), whether in the operating theatre, in the emergency room or also outside the hospital itself. Innovative concepts and very important advances in knowledge have sometimes been obtained in these non-ICU settings.

Dr Baker offers us a second revised and updated edition of his excellent book *Artificial Ventilation: A Basic Clinical Guide*, aimed precisely at this increasingly wide audience of physicians and health professionals who use artificial ventilation. This second edition was eagerly awaited because progress in medicine is rapid and the first edition filled an important educational gap. The recent COVID-19 pandemic illustrated the importance of artificial ventilation for the most severely affected patients, and especially the need for cooperation between emergency physicians, anaesthetists, intensivists and many other specialties to optimally manage patients of varying severity, and sometimes to avoid artificial ventilation whose adverse effects are now better known. I am convinced that this second edition will be as successful as the first. Both experienced physicians and medical students will find in it a compendium of the essential knowledge of this technique, which is so important for more and more patients.

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# Preface to the First Edition

The support of breathing by artificial ventilation has been described for several thousands of years, but a systematic approach to the subject has only developed in recent times. Mechanical artificial ventilation is now a standard part of medical practice in hospital operating theatres and intensive care units (ICU) as well as in emergencies where normal breathing has failed and in the transport of ventilator-dependent patients both within the hospital and between units, sometimes over a range of several thousand miles. This development has not been straightforward. Normal breathing depends on drawing in air to the lungs by creating a partial vacuum inside the chest cavity. From the seventeenth century onwards, artificial ventilation was developed which was the exact reverse of this process with air being forced into the lungs by bellows devices. This was the first mechanical attempt at what is now known as intermittent positive pressure ventilation. During the nineteenth century, this approach fell into disrepute and methods of negative pressure artificial ventilation were developed which sought to imitate natural breathing. This led to the development of the first mechanical cabinet ventilator (or 'iron lung') and several methods of first aid artificial ventilation by manipulation of the arms. Negative pressure artificial ventilation was in turn replaced by intermittent positive pressure ventilation in the middle of the twentieth century with ventilation by bag valve devices and early mechanical ventilation in the ICU from the 1950s onwards. Since that time ventilation of the lungs has become an integral part of both hospital practice where disease or injury causes respiratory failure and emergency medical practice, including cardiopulmonary resuscitation. Although positive pressure artificial ventilation remains common to both prehospital and hospital practice, the means of delivering it in these two settings have become widely separated. The increasing sophistication of computer-controlled ICU ventilators has made their operation very much the work of specialists, both medical and nursing and, in the USA, respiratory therapists. The complexity of the ventilators has been driven by the requirements of ICU physicians and respiratory therapists who increasingly require a form of ventilatory support which matches closely normal physiological respiration and is linked to the patient's own respiratory efforts. Outside the ICU however, many non-specialist medical, paramedical and nursing personnel are

increasingly expected to provide IPPV in emergency settings both in and out of the hospital using more basic portable equipment. This also includes all responders providing ventilation as part of cardiopulmonary resuscitation.

In the emergency setting, emergency ventilation has been provided for over 50 years by the use of hand-operated bag valve devices. The development of portable gas-powered ventilators starting in the 1970s allowed automatic mechanical ventilation in the emergency setting where large and complex ICU ventilators cannot be used. But portable ventilators too have become complex and are often poorly understood by many emergency responders who have preferred using manual ventilation with bag valve devices. Recently, the safety and effectiveness of these devices has been brought into question and has raised the profile of the provision of training in artificial ventilation in emergency, which for many years has been rather neglected compared with other aspects of life support.

In addition to emergency ventilation of individual patients, mass artificial ventilation may be required for casualties of both physical, following earthquakes, and toxic trauma from chemical agent releases, with the use of stockpiled simple ventilators used in non-ICU hospital high dependency units. Equally, mass ventilation may be required for victims of epidemics as the SARS and Ebola outbreaks have demonstrated in recent years. Even in developed countries, the hospital ventilation capabilities may rapidly be overwhelmed and casualties may have to be ventilated with stockpiled portable ventilators by medical and nursing personnel who have little experience in this area. There has been increasing interest among ventilation professionals in the type of ventilator that should be stockpiled in such situations and the training that should be provided for the users.

There is an evident need for improved training and understanding of the principles and practice of artificial ventilation among non-ventilation specialists. The past three decades have seen the publication of a number of excellent and detailed texts on mechanical ventilation, but these have largely been targeted at the hospital specialist and increasingly concern many of the complex modes of ventilation used in the ICU. Conversely, there are few single texts which deal with the basics of airway and ventilation management for those working in prehospital and emergency medicine. This book seeks to fill this gap by providing a basic clinical guide to the principles and practice of artificial ventilation, both manual and mechanical. It covers the development of artificial ventilation through the ages and the essential anatomy and physiology behind it. Non-mechanical methods of ventilation, rediscovered with the development of cardiopulmonary resuscitation in the middle of the last century, are considered together with a discussion of the use and limitations of manual ventilation devices. Mechanical ventilation is approached essentially from the standpoint of portable pneumatic ventilators which have become widely used in situations where non-specialists are involved. These devices were also used in the transport of ventilator-dependent patients but have been replaced by more complex electronically controlled ventilators in recent years. There is now an increasingly wide range of portable ventilators available on the market which makes the decision of which ventilator to buy and use more difficult. Several good comparison studies have been published, and these are discussed to give the reader an idea of what is

available. The use of portable ventilators in emergency and transport ventilation is considered together with special situations such as the resuscitation and ventilation of neonates and infants, where non-specialists may find themselves having to respond in the absence of specialist care. Ventilation in difficult settings, where equipment and support services may be limited, is also considered together with the important topic of the provision of mass ventilation for disaster and epidemics.

The literature available about artificial ventilation is very extensive, and in keeping with the simple approach adopted in this text, detailed referencing has been avoided. Instead, each chapter contains suggestions for further reading in the form of texts and key articles and reviews which are likely to be of interest to the non-specialist. Where necessary, important primary sources have also been cited, as well as the latest guidelines on ventilation for basic and advanced life support published in 2015 by the International Liaison Committee for Resuscitation. These are listed as suggestions for further reading at the end of each chapter.

It would be impossible within the scope of this text to cover all the many mechanical ventilators for emergency and transport ventilation that are currently on the market so I have used a selection of representative devices to illustrate the types of ventilator currently available. Comparative details of a number of portable ventilators are given in two appendices, which are designed to help readers choose which ventilator will be suitable for their requirements. In any book of this size, there will inevitably be omissions, but the interested reader who may require more detailed information is referred to more detailed works which are readily available and are listed in the suggestions for further reading.

I would like to thank the various authors and publishers who have given permission for reproduction of many of the illustrations in the book. I am also very grateful to the Research and Development section of Pneupac Ventilation (Smiths Medical International, Luton, UK) with whom I have worked for many years and who have provided much technical data. I would also like to acknowledge the support I have received from my editor Liz Pope at Springer Verlag without whose help and encouragement this publication would not have been possible. Finally, my thanks go to my wife and anaesthetic colleague Dr Marian Barry for her invaluable help with reading the proofs.

I hope that this basic guide will be of some help to all those medical, paramedical and nursing personnel who are involved in providing artificial ventilation and to whom it is dedicated.

David J. Baker

## Preface to the Second Edition

The intention of the first edition of this book was to provide ambulance and non-specialist hospital personnel with a simple account of the development of artificial ventilation (AV), together with the essential anatomy and physiology behind it and the design, function and use of mechanical ventilators. From the feedback I have received personally and from that contained in articles written by those who have been kind enough to review the original text, this objective seems to have been achieved.

However, the world in 2020 is markedly different from that of 2016, when the first edition appeared, following the pandemic of the coronavirus SARS-CoV-2. Not only has the COVID-19 disease it causes and in many cases acute respiratory distress syndrome (ARDS) led to intense study of ventilation strategies in intensive care units of hospitals, there is now a heightened interest in mechanical ventilation among medical, paramedical and nursing personnel and the general public alike. It is likely that the lessons learned from the provision of artificial ventilation for COVID-19 patients will have an impact on other areas of clinical practice with an increasing realisation of the need to protect the fragile lungs from the essentially non-natural process of positive pressure ventilation.

Although the objectives of this new edition remain the same as the first, I felt it important to include a new chapter to explain artificial ventilation in the intensive care unit (ICU) and ARDS to the intended readership, given the many questions that have been raised during the COVID-19 pandemic. While most of the often complex adaptive ventilation modes used in the ICU will not, at present, be appropriate to providing AV and the prehospital and general hospital settings, it is likely that these will have an impact on the design and use of portable ventilators in the years to come.

The text in this edition has been revised to incorporate corrections and suggestions provided by several reviewers of the first edition to whom I am most grateful. In addition, I would like to thank Professor Bruno Riou, MD, PhD, Dean of the Faculty of Medicine, Sorbonne Université, Department of Emergency Medicine and Surgery, Hôpital Pitié-Salpêtrière, Assistance Publique-Hôpitaux de Paris, Paris, France, for kindly providing a foreword to the new edition and Dr Michael Nurok,

MB ChB, PhD, FCCM, Director, Cardiac Surgery ICU, Smidt Heart Institute, Cedars-Sinai Medical Center, Los Angeles, California, for his invaluable help and suggestions for the new ICU chapter. My thanks also go to my wife and anaesthetic colleague Dr Marian Barry for again patiently checking the proofs.

This book is dedicated, as it was before to all those who are involved in providing artificial ventilation and particularly to all those who have given their lives caring for those affected by the COVID-19 pandemic.

Souillac, France

David J. Baker

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# Abbreviations

ACV	Assist control ventilation
AHA	American Heart Association
ATP	Adenosine triphosphate
ATV	Automatic transport ventilator (USA)
ARDS	Acute respiratory distress syndrome
ARF	Acute respiratory failure
AV	Artificial ventilation
Bi-PAP	Bilevel positive airway pressure
BVM	Bag-valve mask
C	Compliance (V/P)
CMV	Controlled mandatory ventilation
cm H <sub>2</sub> O	Pressure measured in cm of water
COAD	Chronic obstructive airways disease
CPAP	Continuous positive airway pressure
CPR	Cardiopulmonary resuscitation
CRF	Chronic respiratory failure
EAR	Expired air resuscitation
ERC	European Resuscitation Council
ERV	Expiratory reserve volume
ETT	Endotracheal tube
EV	Emergency ventilator/ventilation
F	Frequency
FRC	Functional residual capacity
Hb	Haemoglobin
HME	Heat moisture exchanger (filter)
IC	Inspiratory capacity
ICU	Intensive care unit
ILCOR	International Liaison Committee on Resuscitation
IRV	Inspiratory reserve volume
kPa	Kilopascal
LMA	Laryngeal mask airway

mmHg	Pressure measured in mm of mercury
MRI	Magnetic resonance imaging
MV	Minute volume
P	Pressure
pAO <sub>2</sub>	Partial pressure of oxygen in the alveoli
paO <sub>2</sub>	Partial pressure of oxygen in arterial blood
pCO <sub>2</sub>	Partial pressure of carbon dioxide
peCO <sub>2</sub>	End tidal partial pressure of carbon dioxide
PEEP	Positive end expiratory pressure
PGPV	Portable gas-powered ventilator
PIP	Patient inspiratory pressure
pO <sub>2</sub>	Partial pressure of oxygen
PSV	Pressure support ventilation
R	Respiratory quotient
RF	Respiratory failure
ROSC	Return to spontaneous circulation
RV	Residual volume
SARS	Severe acute respiratory syndrome
SARSCoV2	Severe acute respiratory syndrome coronavirus 2
SIMV	Synchronised intermittent mandatory ventilation
SMMV	Synchronised mandatory minute volume
SV	Spontaneous ventilation
TLC	Total lung capacity
V	Volume
VC	Vital capacity
VILI	Ventilator-induced lung injury
V <sub>t</sub>	Tidal volume
V/Q	Ventilation–perfusion ratio

# Chapter 1

## A Brief History of Artificial Ventilation



### 1.1 Introduction

Artificial ventilation (AV) of the lungs to replace failed normal breathing goes back to antiquity but it is over the past 300 years that it has developed to the essential position it holds in modern medicine. Now, AV using both simple and highly complex devices is found in all areas of medical practice, from the emergency ambulance through to the sustained life support of a critically—ill patient in an intensive care unit. It is a fundamental part of cardiopulmonary resuscitation (CPR) for primary cardiac arrest and for respiratory failure and arrest which leads to secondary cardiac arrest as well as in the management of asphyxiation from other causes such as carbon monoxide poisoning and drowning. Major trauma may cause respiratory failure or complete arrest and here too AV is an essential part of the clinical management.

Outside the hospital AV may be performed by first aiders using only basic skills and by medical or paramedical personnel with relatively sophisticated equipment. In the hospital setting AV is used on a regular basis as part of general anaesthesia when the patient may require ventilation support as part of balanced anaesthesia where the patient may require muscular paralysis to aid surgery. In this situation the patient cannot breathe of his own accord since the diaphragm and other respiratory muscles are also paralysed by neuromuscular blocking drugs derived originally from the South American arrow poison curare. In the intensive care unit (ICU), AV is used to support or completely replace the breathing of patients who are critically—ill either from infection, trauma or following major operative procedures. Artificial ventilation in all these different settings, both in and out of the hospital follows the same basic principles which are described in later chapters in this book. However, the skill levels and ventilators used in modern clinical practice now cover a very wide range.

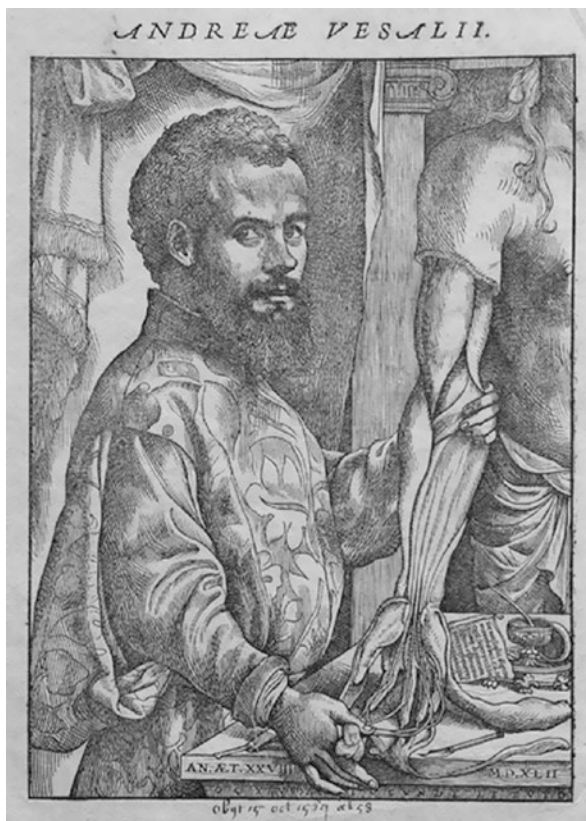
In the early years of resuscitation and anaesthesia simple techniques of AV were used, both inside and outside the hospital. Since the 1960s however hospital ventilators, used in ICU have become increasingly complex depending on piped high pressure gas supplies, electromagnetic valves and microprocessor controls. Over the same time span, there has been a parallel development of portable resuscitator devices and ventilators designed to be used by ambulance services, in the emergency room and in the recovery areas of operating theatres. The situation now is that there are two essentially different classes of mechanical ventilator namely (1) sophisticated devices designed to provide optimal interactive patient respiratory support in the hospital, usually in the ICU (2) simple portable ventilators that are designed to be operated by non—ventilation specialists in a variety of non-ICU and prehospital settings which include disasters and mass ventilation. Over the past decade this division has become more complicated with the development of electronically—controlled complex portable ventilators designed for the transport of ventilator—dependent patients from one location to another, while providing the level of complexity of AV support found on modern ICU ventilators.

This chapter concerns the early development of resuscitation, through the development of manual ventilation devices and the renewed interest in expired air ventilation as part of CPR in the early 1960s, followed by the development of and use of portable pneumatic ventilators which began in the 1970s. Where necessary there will be references to the development of hospital ventilators but, in keeping with the objectives of this book the emphasis will be on manual techniques and portable ventilators intended for use by non-specialist providers of AV. There are many detailed accounts of the history of AV in the literature, some of which are included in the suggestions for further reading at the end of this chapter and readers requiring more detail are referred to these.

## 1.2 The Development of Basic Methods of Artificial Ventilation

Through the ages simple AV has involved blowing air or expired air into the victim's lungs or drawing air into the lungs by a variety of procedures which attempted to expand the thoracic cavity by physical means. Both these approaches had the advantage of requiring little or no equipment. Expired air resuscitation, (EAR), possibly first described in the Old Testament (II King's 4, vs 4–5) relies on the fact that air expired from normal lungs contains sufficient oxygen (18%) to oxygenate the blood of someone who has stopped breathing. EAR has a long history but was developed scientifically with the development of modern cardio-pulmonary resuscitation in the 1960s by James Elam and Peter Safar in the United States. The great Italian anatomist Vesalius (Fig. 1.1) in 1543 made the discovery that the pressure inside the thoracic cavity was negative and that this drew air into the lungs. He opened the chest of a live animal and noted that air rushed in and the lungs collapsed, following

**Fig. 1.1** The sixteenth century anatomist Vesalius



which the heart stopped. In his famous work *De humanis corporis fabrica* (*Concerning the fabric of the body*), published in 1543 he described 'placing a tube in the trachea and blowing into it to re inflate the lungs.' In England in the seventeenth century Robert Hooke repeated Vesalius experiments and in 1667 carried out his own experiments in a dog whose chest had been opened and therefore normal breathing was not possible. Hooke used positive pressure ventilation from a bellows which kept the dog alive and provided the first demonstration that it was the gas entering and leaving the lungs and not the movement of the lungs themselves as though previously.

Interest in AV continued into the eighteenth century and in 1744 Tossach, a Scottish physician, gave an account of the use of EAR on a miner who had suffocated. EAR was used in midwifery at that time but was not adopted by the medical profession nor the public. Positive pressure ventilation continued to develop however. 1767 saw the founding of the Society for the Resuscitation of the Apparently Drowned in Amsterdam. Other societies were formed in Venice and Milan in 1768, along with the Royal Humane Society in London in 1774 Positive pressure ventilation was provided by a modification of the household fireside bellows and versions

**Fig. 1.2** The royal humane society resuscitation bellows 1784 (Reproduced with permission from Cazalaa et al. (2005). Photograph by Jean-Pierre Alonso)



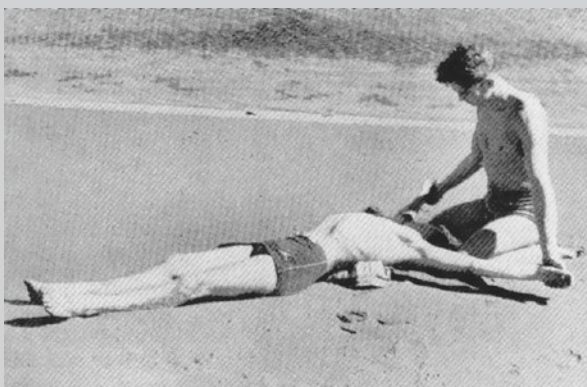
of this were used in Amsterdam and London (Fig. 1.2). In London the surgeon John Hunter developed his own version of the resuscitation bellows which both pumped air into the lungs during inspiration and sucked it out again during expiration.

One major problem faced with this approach to AV was the difficulty establishing a connection between the ventilation device and the patient. This highlighted the importance of establishing a proper airway through which AV could be provided and led to the development of the technique of intubation of the trachea. The development of airway management in relation to AV is considered further in Chap. 5.

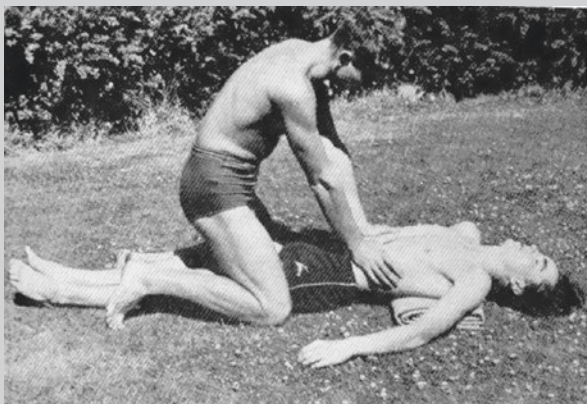
Although shown to be effective, early positive pressure ventilation had its hazards however and in Paris, in 1827 Leroy demonstrated that damage could be caused to the lungs by over-inflation. From that time the bellows technique fell into disrepute and was discontinued. Before the rediscovery of modern positive pressure AV there was more than a century of the use of a number of negative pressure techniques that were introduced after the abandonment of bellows ventilation. Between the 1830s and 1940s more than 70 methods of negative pressure ventilation (where the normal ventilation of the lungs is mimicked by a number of manipulations designed to suck air into the lungs) were described. These involved stretching the chest wall muscles by lifting the arms followed by compression of the thorax or abdomen to aid expiration introduced by Sylvester in 1861 and Howard in 1871 (Box 1.1). By the late 1950s these methods were realised to be totally ineffective in providing adequate ventilation during resuscitation and were discontinued.

**Box 1.1 Manual Negative Pressure AV**

In the latter part of the nineteenth century AV methods involving stretching the arms to promote inspiration and compressing the thorax to help expiration were introduced by Sylvester in 1861 and Howard in 1871. They were later combined to produce the Holgar Neilson method widely taught to first aiders in the mid twentieth century.



Inspiration



Expiration



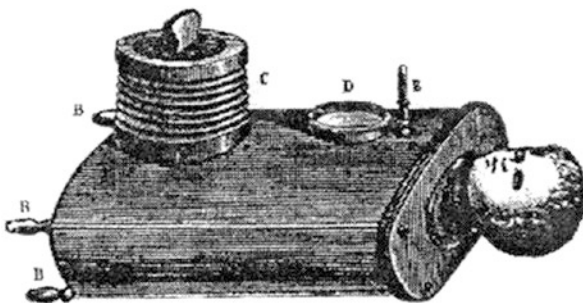
### 1.3 The Development of Mechanical Negative Pressure Ventilation

From the late nineteenth century through to the middle of the twentieth, mechanical AV was provided by ventilators that used the principle of negative pressure ventilation, where a partial vacuum was created around the patient in order to suck air into the lungs from outside. Such devices were called ‘cabinet ventilators’ or more popularly ‘iron lungs.’ The Frenchman Woillez described the first negative pressure machine in 1876 (Fig. 1.3). This was called the spiraphore and consisted of a metal chamber around the body with the patient’s head sealed thorough a rubber flange at one end of the chamber. The ventilator was powered manually by a lever connected to a flexible diaphragm at the other end of the box. The device had a number of disadvantages due to being operated manually and with leaks occurring around the neck seal which meant that the negative pressure in the chamber was ineffective. However the idea of cabinet ventilation crossed the Atlantic and in 1929 Drinker and McCann in Boston built an electrically—driven device. These workers were copied by others. The iron lung became a feature of hospital practice and was used extensively in the management of poliomyelitis for nearly 30 years. Figure 1.4 shows an iron lung ward in California during the polio epidemic of 1953. However such machines were in short supply for mass ventilation and it was the need for an alternative in Copenhagen in 1952 that led to the development of modern positive pressure ventilation.

### 1.4 The Return of Expired Air Resuscitation as Part of Cardio-Pulmonary Life Support

James Elam, in late 1950s against resistance from first aid organisations, showed that EAR with effective airway control could provide normal levels of oxygen and carbon dioxide following apnoea for long periods. The Austrian Peter Safar and his colleagues, working in Pittsburgh in 1960 added head tilt and chin lift for Elam’s EAR technique making it possible for a first aider to keep the airway open and

**Fig. 1.3** The Woillez Spiraphore 1876  
(Reproduced with permission from Cousin (2005))







**Fig. 1.4** A cabinet ventilator ward in California during the 1953 polio epidemic

provide artificial respiration without the aid of any equipment. At the same time, Koevenhoeven, Jude and Knickerbocker developed the idea of chest compression combined with EAR to produce the first systematic approach to cardiopulmonary resuscitation (CPR). Chest compression alone had been suggested earlier as a method of AV but was shown not to provide an adequate tidal volume. With the combination of expired air ventilation the essential airway, breathing and circulation (ABC) management of life support was born and has continued to the present day. To improve ventilation further, it was clear that other techniques would be required. The major driving force in this area came from the development of assisted ventilation in anaesthesia which had been used since the 1930s.

## **1.5 The Development of Airway Management in Relation to Artificial Ventilation**

### ***1.5.1 Introduction***

The human airway is not stable and requires support during unconsciousness and when there are life—threatening blockages. This fact had been realised since the birth of medicine but it was with the development of methods of positive pressure

ventilation, starting in the eighteenth century that the importance of airway management during AV was truly recognised.

The history of airway management is complex and goes back many centuries but today endotracheal intubation is recognised as being the most effective way to protect the airway and to ensure adequate ventilation during AV. Many forms of endotracheal tube have been produced in the last 100 years and accounts of the history are often clouded by linguistic and national considerations. While development in the United Kingdom and the United States was linked by a common language in journals that were widely read on both sides of the Atlantic the important developments in airway management in France and Germany often went unnoticed by workers who only read and spoke English. Recently the history of the development of airways in France has had a wider readership through the publication of accounts in English.

For a full account of the history of airway management the reader is referred to specialist articles and texts. This section considers the development of airway management in direct relation to the development of AV, which could not have progressed without it.

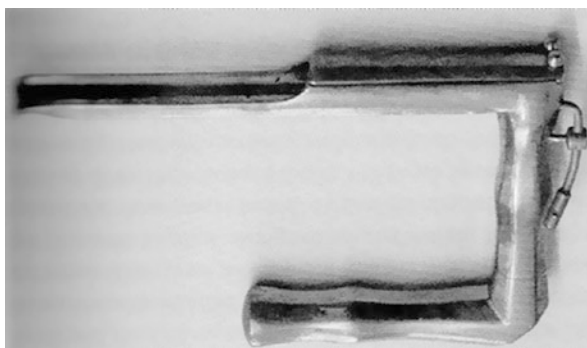
### ***1.5.2 Driving Forces in the Development of Endotracheal Intubation***

Placing a tube in the windpipe to overcome obstruction to breathing has been known since antiquity but it was the animal studies on resuscitation in the seventeenth century by Robert Hooke and others that revived interest in its use. Although the significance of respiration and the need to support it was emerging from animal studies, in human medicine the use of tracheal intubation was driven by common emergencies affecting the respiratory system. The urgent requirement for a clear airway was recognised by the early eighteenth century. In England the surgeon John Hunter realising that AV could not be effective without a clear passage to the lungs carried out early experiments with AV from a basic fire bellows on intubated dogs. However, the early drive to tracheal intubation did not come from AV but from common life—threatening respiratory conditions that obstructed the airways. The French obstetrician Chaussier (1746–1828) described the use of a metal tube to be inserted into neonates, guided by the fingers. The need for maintaining an open airway because of disease was further recognised by Bouchet in 1858 who used intubation to overcome croup in children. Several other French physicians described intubation through the nineteenth century, despite the fact that positive pressure AV had been abandoned as unsafe at that time. O' Dwyer, working in New York published a paper in 1885 on the intubation of the larynx for cases of diphtheria which was to be of major influence in the development of modern intubation. This worker was however unaware of the work of Bouchet in France.

Trauma provided another drive to the development of intubation. Charles Kite, in 1788 in an essay on resuscitation described placing a tube in the nose to support respiration in the case of pharyngeal blockage. Currey, in 1815 described intubation techniques for casualties trapped in mines and caves. The progress towards intubation continued into the twentieth century. Intubation became increasingly necessary in the management of anaesthesia for the trauma cases during the First World War. Early tubes were made of metal but the first flexible tube fitted with a cuff to ensure a good seal within the trachea was invented as early as 1898. In 1919 the English anaesthetists Magill and Rowbotham described the forerunner of the modern endotracheal tube (ETT). This had both an entry tube for fresh gas and an exit tube for expired air. In 1938 Magill concluded that a single tube made of red rubber to function during both inspiration and expiration would be better. These ETT continued to be standard equipment in anaesthesia until the 1970s when they were gradually replaced by plastic disposable tubes developed by the Portex company, founded in Portland Place London by the dentist SA Leader. These tubes in a variety of forms are in use in anaesthesia and emergency medicine to the present day.

The progress of intubation in the twentieth century was greatly helped by the development of laryngoscopy—the ability to view the larynx and to insert a tube under visual control rather than guided by the operator's fingers (Fig. 1.5). Babbington in England had described indirect laryngoscopy in 1829 but the first direct laryngoscope was developed by the American Chevalier Jackson in 1910. This was made possible by the ability to illuminate the larynx by the use of a small electric light bulb, a development first described by Baratoux in France in 1883. Before this time the only option for illumination during any form of endoscopy was to use a naked flame built into the instrument. While this was possible in early urethroscopy it was not practical for airway management. Following the First World War laryngoscopy developed and became widely accepted in anaesthesia with the development of a straight blade device by Magill between 1923 and 1928 and later with the familiar curved blade Mackintosh laryngoscope in 1943.

**Fig. 1.5** The Chevalier Jackson Laryngoscope 1910 (Author's collection)



### ***1.5.3 The Development of Other Means of Airway Control***

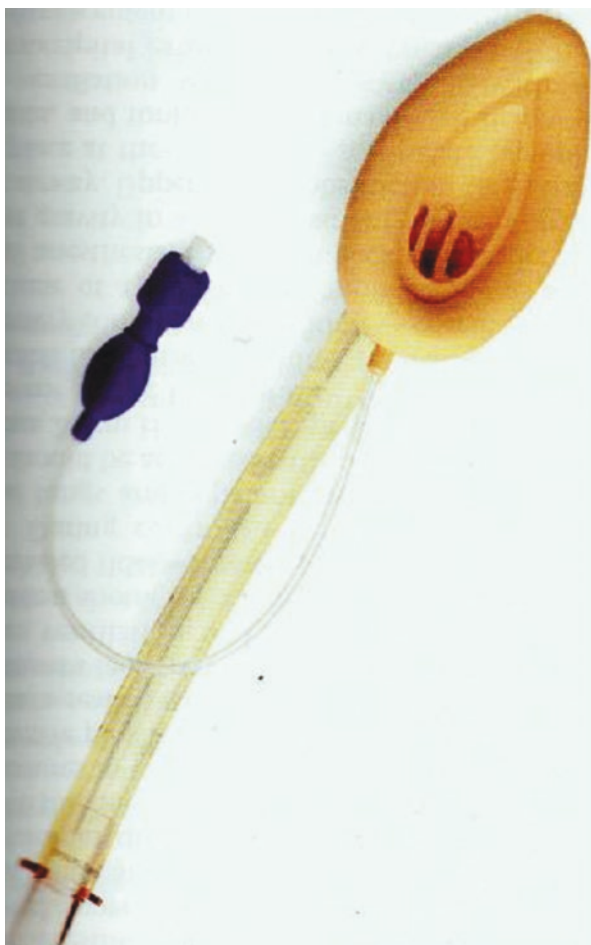
With the development of general anaesthesia in the nineteenth century there was a need to keep the airway open in an unconscious patient. In 1873 the German anaesthetists Heiberg described the now classical technique of jaw thrust, while Howard in England described head tilt and chin lift while delivering the anaesthetic through a facial mask. At a time when most anaesthesia was being given in this way the development of oropharyngeal airways such as that of the Americans Guedel and Waters in the early 1930s was a major step forward.

However, by the early 1980s the only alternative way of ventilating a patient who could not be intubated was to use a pharyngeal mask and airway. This was not always the best option for patients with a full stomach in the case of emergency anaesthesia who were at risk of vomiting and inhalation. The answer to this problem was provided by the English anaesthetist Archie Brain. A cuffed oropharyngeal airway had been developed in the United States by Shipway in 1935 but interest in this type of device which could protect the airway against reflux from the oesophagus was not revived until Brain's work in the late 1970s. He noticed that the shape of the pharynx, revealed by a plaster cast of a cadaver resembled an early nasal anaesthetic mask, developed by Goldman for use in dental anaesthesia. Brain made his own prototype laryngeal mask airway (LMA) as it was to become known, by attaching a Goldman mask to a Portex ETT. After working for a number of years on prototypes he produced a mask that effectively sat over the larynx in the way the traditional mask sits over the mouth and nose (Fig. 1.6). In 1981 Brain performed the first blind insertion of an LMA followed by effective mechanical ventilation. His paper on the subject, published in 1983 was largely ignored but he persevered with this device and by the mid-90s it was widely adopted by many hospitals in the UK and the rest of the world. Its advantages in being able to provide a secure airway (with limitations) without the need for intubation was of considerable importance for non-specialists who were required to ventilate patients but who had very little continuing experience of intubation. Despite early concerns about the LMA not providing an effective seal during positive pressure ventilation it is now recognised by some emergency services, particularly paramedical as being the airway management of choice for emergency ventilation in difficult circumstances.

## **1.6 The Modern Development of Positive Pressure Ventilation**

We have seen that the support of breathing from the middle nineteenth century onwards both in and out of hospital relied on the use of methods that would support natural breathing by creating a negative pressure in the chest cavity. Negative pressure mechanical ventilation using an iron lung had a number of disadvantages which were: (1) the machine was very large and expensive to purchase and maintain (2) access to the patient for standard nursing care was very limited (3) the patient had

**Fig. 1.6** An early Laryngeal Mask Airway, 1981 (Author's collection)



to synchronise his swallowing and phonation with the machine (4) the neck seal had to be tight and was often uncomfortable and could cause ulceration of the skin (5) if the patient had paralysis of the laryngeal or pharyngeal muscles there was a risk of regurgitation and inhalation. This could be managed by a tracheostomy but looking after the tube with the patient inside the chamber was very difficult. These problems and the realisation of the ineffectiveness of manual methods of AV led to the re-introduction of positive pressure ventilation as a standard procedure by the 1950s. The stages in development can be summarised as follows:

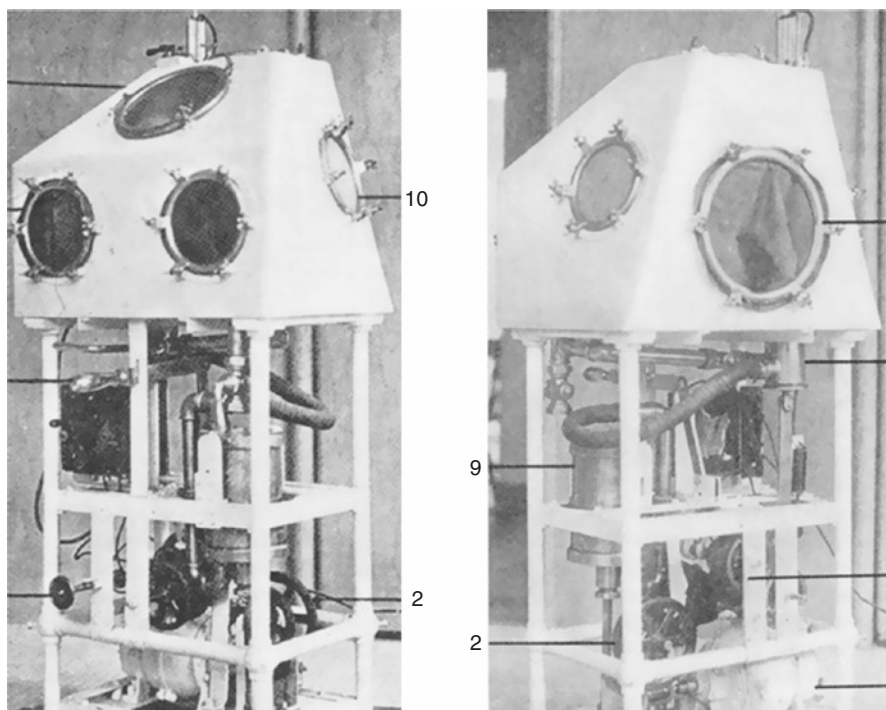
1. The first operations which opened the chest cavity were performed in the USA towards the end of the nineteenth century. Because the normal negative pressure seal in the chest had been broken there was therefore a need to provide internal lung pressure to keep the lungs open and to ensure adequate ventilation.
2. In Germany in 1907 the Dräger company produced the first mechanical ventilator, the Dräger Pulmoflator (Fig. 1.7). This interesting device, discussed in more

detail below incorporated many of the principles that were to be used in pneumatic ventilation 60 years later. It was designed to be used to provide AV in mines (Dräger was a company specialising in mining industry support) but inexplicably the medical profession largely ignored it.

**Fig. 1.7** The Dräger Pulmotor Artificial Ventilator 1907 (Reproduced with permission from Cazalaa et al. (2005). Photograph by Jean-Pierre Alonso)







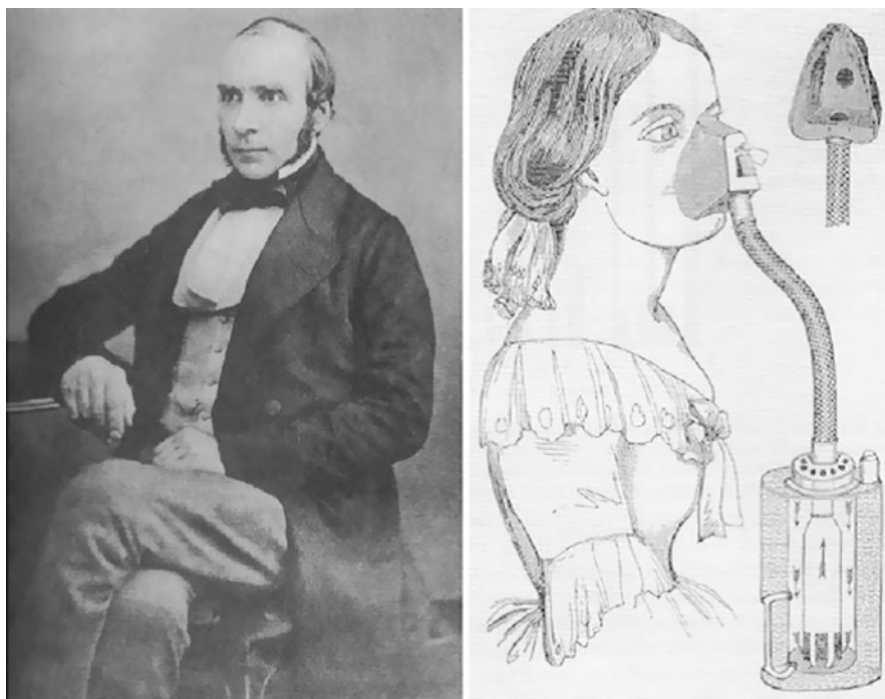
**Fig. 1.8** The Green and Janeway Headbox circa 1900

3. In 1910 Laeven and Sievers developed an electrically driven piston ventilator for the management of pulmonary embolectomy (a clot of blood in the main vessels of the lungs).
4. Green and Janeway in New York produced artificial positive pressure ventilation via a box placed around the patient's head (Fig. 1.8) These innovators coined the term 'controlled ventilation' since their machine could control the rate and volume of each delivered breath and could also apply positive pressure during expiration—a technique that would become a standard medical procedure in the later part of the twentieth century. The concept of controlled ventilation was the root from which all ventilation would develop in the latter part of the twentieth century.
5. In 1927 the American Dennis Jackson used an anaesthetic machine combined with a mechanical ventilator for the first time. However, it was used only in veterinary medicine and it aroused no interest in human practice. Jackson was the first to continue the studies of positive pressure ventilation that had begun with the work of Vesalius nearly 400 years earlier but were discontinued at the beginning of the nineteenth century.
6. Meanwhile, controlled ventilation made its first re-appearance in human anaesthesia with the work of the anaesthetists Arthur Guedel (who invented the curved pharyngeal airway that is still a standard device to the present day) and David

Treeweek. These innovators used their machine for thoracic anaesthesia from 1934 onwards. There were also developments in Sweden to produce mechanical controlled ventilation with the production of the Spiropulsator ventilator. IPPV had been suggested as early as 1916 in Sweden by Giertz but his publication was in Swedish and went unnoticed. Despite all these interesting developments on the production of mechanical ventilators most anaesthetists continued to support ventilation during general anaesthesia by squeezing the reservoir bag of the anaesthetic machine. But this technique would be the forerunner of the modern self-reforming bag (the 'Ambu' bag) and of modern intermittent pressure ventilation).

## 1.7 The Development of Positive Pressure Ventilation in General Anaesthesia

Early anaesthesia depended on the patient inhaling a volatile anaesthetic agent at atmospheric pressure. Figure 1.9 shows a typical apparatus developed by the English pioneer anaesthetist John Snow. This type of inhalation anaesthesia was used in



**Fig. 1.9** A mid-nineteenth century inhalational anaesthesia apparatus (*right*) developed by John Snow (*left*) (Reproduced with permission from Cazalaa et al. (2005). Photograph by Jean-Pierre Alonso)



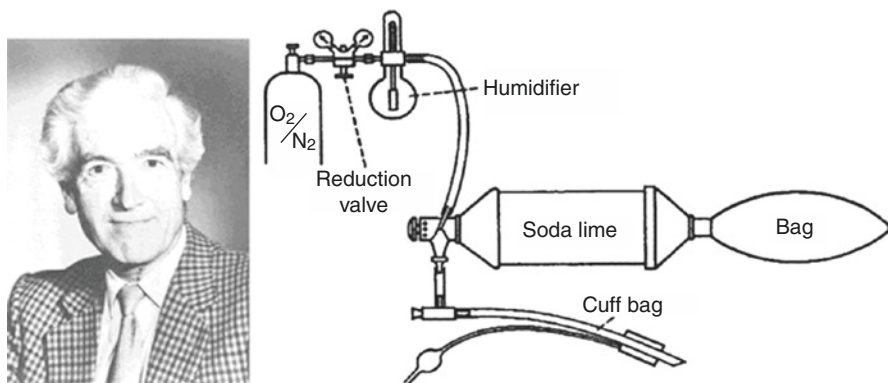
hospitals well into the twentieth century. Gradually however it became necessary to mix oxygen and nitrous oxide under positive pressure from cylinders to take and to deliver the anaesthetic agent, such as ether to the patient. This led to the development of the so-called anaesthetic ‘machine’ which was essentially composed of gas cylinders, flowmeters and a vaporiser. An integral part of this device was the reservoir bag. When the patient is breathing spontaneously the bag (called a Boyle bag after the British Anaesthetist Boyle) acts as a reservoir and its volume decreases. Anaesthetists found that they could assist the patient’s own respiratory efforts by squeezing the bag while the expiratory valve in the pipe leading to the patient (known in anaesthetic terms as a ‘circuit’) through which the patient exhales was partially closed. This gentle squeezing of the bag became known as ‘assisted ventilation.’ Gradually anaesthetists realised that controlled ventilation, where the AV took over completely from the patient’s own efforts was a more effective way of ventilating the lungs.

The development of muscle relaxant agents for use in anaesthesia provided another major stimulus for the development of positive pressure ventilation. 1942 saw the first use of curare (known for many centuries in South America as a paralysing arrow poison) to provide relaxation of the muscles to aid surgery. Previously this could only be achieved by deep inhalational anaesthesia from which the patient would take many hours to recover consciousness. The rationale for the use of curare was to relax the abdominal muscles. However, paralysing these also paralysed the respiratory muscles, first of the chest wall and then the diaphragm. Thus AV was necessary to maintain life during an operation.

The use of curare and synthetic drugs with the same action gradually become standard practice in anaesthesia during the second part of the twentieth century and so positive pressure ventilation also became standard practice. Another key development in anaesthesia to provide manual positive pressure ventilation both in the operating theatre and in emergency resuscitation outside the hospital was to modify the Boyle bag so that it would function without an internal positive pressure flow of gas. This development occurred after the first use of anaesthetic equipment for non-anaesthetic purposes during the Copenhagen polio epidemic of 1952. This was the beginning of the standard use of AV for life support in conditions that produce partial or complete respiratory failure.

## 1.8 The 1950s and the Start of Intensive Care Units

The requirement for mass ventilation in Copenhagen during the 1952 polio epidemic overwhelmed the supply of cabinet ventilators available. The anaesthetist Bjorn Ibsen had the idea of using anaesthetic circuits containing a Boyle bag to provide intermittent positive pressure ventilation. The bag was kept inflated by a positive pressure of gas within the circuit and could be squeezed by hand to provide inflation (Fig. 1.10). Later the bag would be modified to become self-reforming so that it could be used independently of an anaesthetic machine.



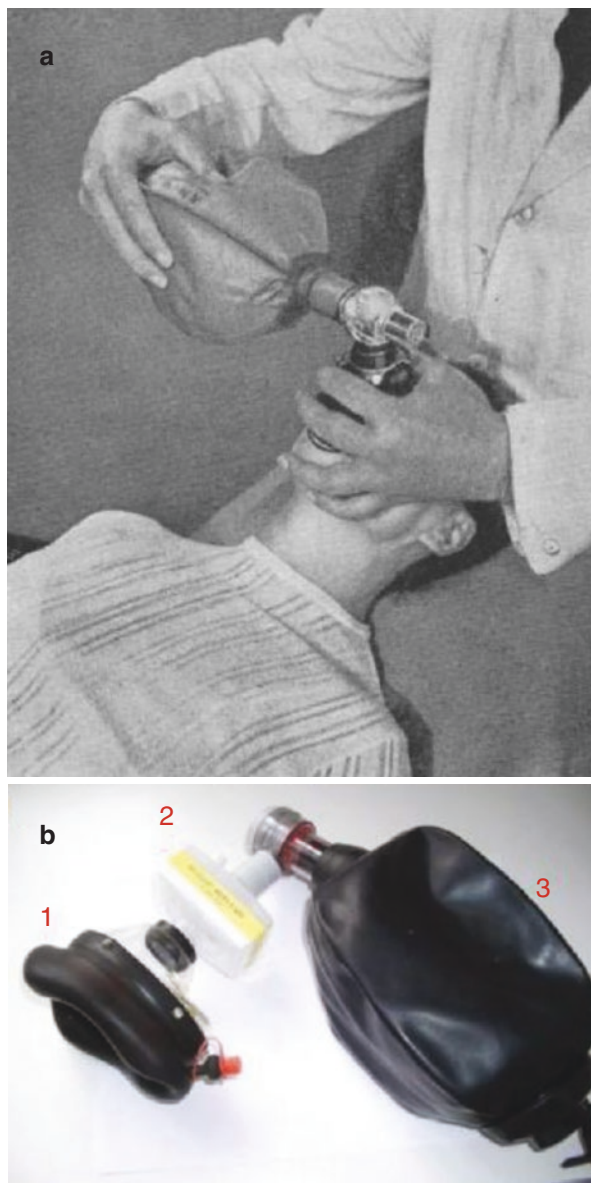
**Fig. 1.10** The Danish anaesthesiologist Bjorn Ibsen and a manual ventilation apparatus used in the Copenhagen polio epidemic in 1953 (Author's collection)

The bag used by Ibsen had to be kept inflated by a constant flow of gas, which was normal in anaesthesia. The Danish anaesthetist Henning Ruben modified the bag by placing springs inside which would allow the bag to reform after squeezing. Apart from the self-reforming bag Henning Ruben also invented the Ruben non-return valve which allowed the escape of expired carbon dioxide to the surrounding air. Previously, as in the case of Ibsen, patients had to be ventilated with a circuit containing a soda lime canister to absorb the gas. The incorporation of a non-return valve led to the development of modern self-reforming bag, the first example of which was the Ambu bag in the late 1950s. This device and others like it have been widely used in anaesthetic rooms and in both hospital and prehospital emergency medicine since that time. In conjunction with a pharyngeal mask, the bag-valve-mask is probably the most widely—used device to provide positive pressure ventilation in the world today. Figure 1.11 shows early and modern versions of this device.

### ***1.8.1 The Use of the Self-Reforming Bag in Emergency Medicine***

The development of the self-reforming bag to provide manual positive pressure ventilation using the surrounding air changed the world of emergency medical care and allowed reliable and effective AV to be provided for both medical and traumatic surgical emergencies outside the hospital. The Ambu bag quickly became incorporated into emergency medical practice along with the use of expired air resuscitation. These two methods of AV remained the only means of providing AV outside the hospital until the development of portable gas—powered ventilators a decade later.

**Fig. 1.11** (a) An early AMBU bag—valve device  
(b) a later version  
(Author's collection and courtesy of AMBU, Copenhagen)



The self-reforming bag has become standard equipment in both the prehospital and hospital settings where it is used for a number of purposes by medical, paramedical and nursing staff. In some countries, notably the USA it is standard equipment for paramedics in the prehospital setting. Recently however, the effectiveness and safety of the device have been questioned. These concerns are discussed further in Chap. 5.

## 1.9 The Development of Portable Mechanical Ventilators

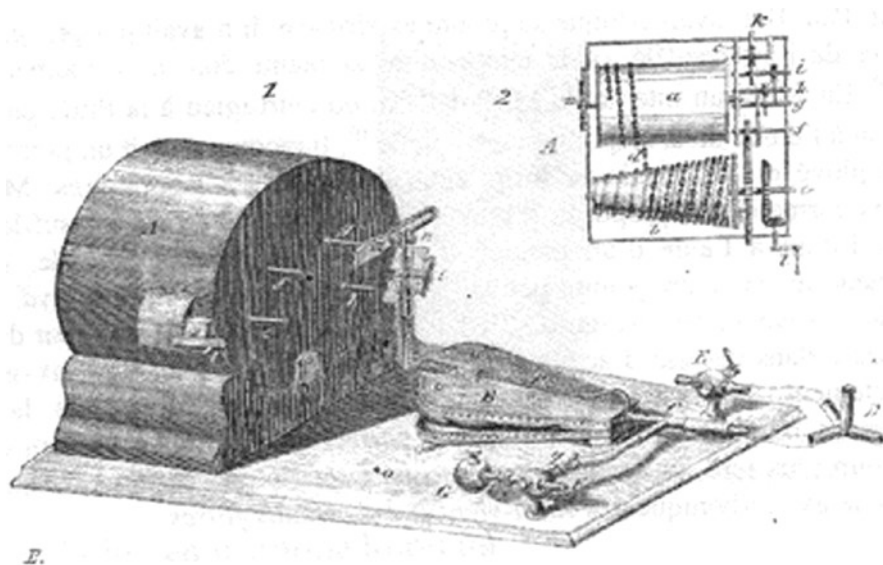
### 1.9.1 *Early Development*

Following the polio epidemic of the early 1950s and with the routine use of muscle relaxants in general anaesthesia there was a surge in the development of large and increasingly sophisticated mechanical ventilators for use in the operating theatre and the intensive care unit. But at the same, beginning in the 1970s there was a development of small portable ventilators that could be used to provide automatic ventilation in emergencies and for the transport of ventilator—dependent patients, both inside and outside the hospital. The development of ICU and portable ventilators took different pathways which have only converged in recent years with the production of small portable ventilators which are computer—controlled.

The development of portable ventilators, initially used for resuscitation and later for transport of ventilator—dependent patients started earlier than is usually realised and before the appearance of large mechanical ventilators in the 1950s. The first portable ventilator, designed for use in mines, the Pulmotor (Fig. 1.7) was designed by the German, Heinrich Dräger in 1907. In his publication ‘The Development of the Pulmotor’ Dräger documented his ideas about developing a ventilator. He described a new technology ‘for blowing fresh air or oxygen into the lungs’. In 1907 this device was patented and was used successfully for many years in the mining industry. Despite the innovations of the Dräger family in producing a portable gas—powered ventilator prototype before the First World War the concept of portable mechanical ventilation for resuscitation was slow to catch—on with the medical profession. Several decades were to elapse before the concept was taken up again.

France was among the European countries after the Second World War to develop mechanical ventilation in a systematic way. This followed the progress in developing insufflation devices by the great French physiologists in the nineteenth century and early work on negative pressure ventilation which lead to the development of the famous ‘iron lung.’ The modified bellows ventilator shown in Fig. 1.12 was developed by the great French physiologist Claude Bernard and used for his extensive animal experiments using curare to keep the animal from dying of respiratory failure (this was something that would only be rediscovered in the medical context in the 1940s).

However, by the early 1950s improvements in mechanical ventilation had developed to such an extent that a classification of types of ventilator was possible. This was driven by the French anaesthetists of the time, led notably by Maurice Cara. At the time anaesthesia in France lagged considerably behind developments in the English—speaking countries but the interest in AV was considerable. It was realised by Cara that AV could be classified according to the way the gas was being delivered to the patient. The earliest classification identified (1) ventilation with a fixed frequency, termed volume release ventilation. This related to a ventilation mode where there was a release of excess gas once the desired volume of gas for each respiration



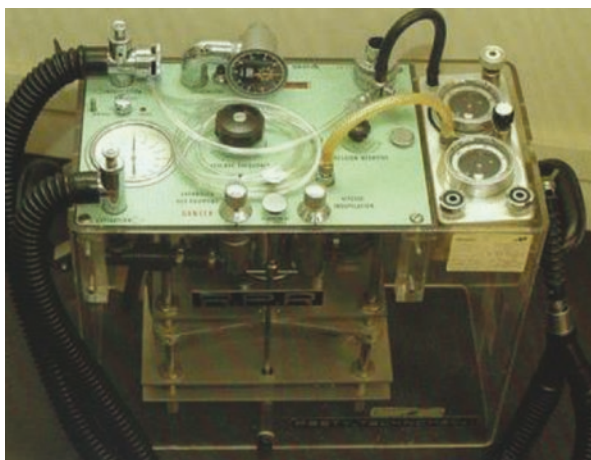
**Fig. 1.12** Claude Bernard's automated ventilation bellows (Reproduced with permission from Cousin (2005))

had been delivered. This covered what would now be termed as 'volume generators' (see Chap. 6). The other broad classification of ventilation was 'pressure generators' later called pressure targeted ventilation. This was when a ventilator changed from inspiration to expiration after a set pressure inside the airways has been achieved. Even at that time it was realised that pressure support ventilation could not guarantee a set volume of gas to be delivered to the patient when the lung characteristics of resistance and distensibility had altered and the mode was largely used for supporting existing patient respiratory efforts rather than replacing them. Thus pressure support ventilation found an early use in the home at that time. It had no place in anaesthesia but as the distinguished French anaesthetist and historian Marie Therese Cousin has pointed out that it was 'found in that setting rather too often' largely as a consequence of their attractive price.

### 1.9.2 The French RPR Ventilator

France was the birthplace of what might be regarded as the first modern portable pneumatic ventilator called the RPR ventilator after the inventors Rosenstiel, Pesty and Richard. This machine, shown in Fig. 1.13 was developed at a time after the second World War when mechanical ventilation was provided by bulky, heavy and expensive ventilators such as the Engstrom (Fig. 1.14) which could not be operated

**Fig. 1.13** The French RPR portable ventilator' 1955 (Reproduced with permission from Cazalaa et al. (2005). Photograph by Jean–Pierre Alonso)



**Fig. 1.14** The Engstrom ventilator 1954: an early example of a hospital mechanical ventilator (Reproduced with permission from Cazalaa et al. (2005). Photograph by Jean–Pierre Alonso)





outside the hospital. The RPR ventilator was designed to provide AV when transporting patients between one hospital ventilator and another. It was designed and produced by the French engineer Rayond Pesty who worked in collaboration with a French navy physician Rosenstiel. The device was a non-rebreathing volume generator and in this respect it was the forerunner of most later gas—powered ventilators. It was powered by compressed gas from a cylinder. The AV it produced depended on two factors (1) the flow of gas and (2) a regulated respiratory pause at the end of inspiration. The ventilation provided in 1 min (the minute volume) was measured using a built-in spirometer. The RPR ventilator was a great success in France following its introduction in 1955 and remained in use until the 1990s. Modifications of the device could deliver small tidal volumes at high frequencies and a version was later developed to provide the first mechanical ventilator for use with neonates and infants.

### 1.9.3 The Development of Portable Ventilators After 1960

Although a small ventilator had been produced by the American aviator Forrest Bird in the USA (at the request of the US Armed Forces) in 1956 (the later development of small portable mechanical ventilators took place largely in Europe), at a time when there was a steady development of large ventilators for use in the ICU in hospital. While these become increasingly complex the development of portable ventilators followed a different line of evolution and remained essentially simple in control and operation. From the outset they were designed for use by non-specialists, including primary emergency responders, while the hospital devices became increasingly the domain of ventilation specialists.

The first pneumatic ventilator specifically—designed for use in emergency by non-specialists was the original Pneupac with the production in the late 1970s of the 2R ventilator shown in Fig. 1.15. which was developed specifically for use in

**Fig. 1.15** The Pneupac 2R ventilator 1978  
(Photograph courtesy of Pneupac Ventilation Smiths Medical International, Luton, United Kingdom)



cardiopulmonary resuscitation. This device was developed by the English physicist and engineer Norman Jones, in collaboration with the Hungarian engineer George Weiss who had an industrial interest in pneumatically operated devices such as automatic doors. Working on an early prototype which had been invented by the English anaesthetist Geoffrey Burchell in the late 1960s, Jones produced a radical new design which was, unlike its predecessor a volume rather than a pressure generator. It was realised early on in the field of resuscitation that pressure generators are not suitable for use in emergency ventilation where the lung stiffness and airway resistance may be altered (see Chap. 6). At the heart of the new device was an ingenious pneumatic timing device which depended on the flow of gas through a restrictor valve into a piston. This pneumatic oscillator remains the heart of pneumatic ventilators to the present day. The immediate attraction of the first Pneupac ventilator was the fact that tidal volume and frequency were controlled by a single control which could be set quickly to provide ventilation for a range of patients ranging from small children to large adults. Because of its strong design the 2R was employed not only in emergency ventilation but also in battlefield anaesthesia and was used successfully in this way by naval anaesthetists during the Falklands War in 1982.

#### ***1.9.4 The Invention of the Demand Valve: Interactive Ventilation Introduced into the Prehospital Emergency Setting***

One major disadvantage of early ventilators, both hospital and portable was their lack of interactivity with the patient's own breathing efforts if there was only partial respiratory failure. In complete respiratory failure where breathing had stopped completely there was no problem in providing IPPV but if the patient retained some of his own breathing efforts these could work against the support being provided by the ventilator—a condition termed 'fighting the ventilator.' This was usually overcome by suppressing the partial respiratory efforts by the use of opioid drugs supplemented in the case of the ICU by muscle relaxant drugs such as curare. Gradually manufacturers began to make the ventilators more interactive with the patient. This was particularly true in hospital ventilation after the period covered by this chapter, where ventilators have become potentially totally interactive with the patient and can analyse each breath by computer to work out what the next breath should be and when it should be delivered.

In the increasingly divergent world of portable ventilation developments took place that could synchronise the work of the ventilator with the patient's own respiratory efforts. The results were modes such as Synchronised Intermittent Mandatory Ventilation (SIMV) and Synchronised Mandatory Minute Volume (SMMV) which are discussed further in Chap. 6. These synchronised modes became possible with the development of the demand valve, a device that could detect a breath from the



patient, usually by measuring the reduction in pressure at the point of delivery of the AV (i.e. the ETT). Demand valve technology was pioneered in the 1980s by the Pneupac company in the UK and radically changed the use of portable gas powered ventilators.

## 1.10 Adoption of the Use of Portable Ventilators

The previous discussion shows that portable pneumatic ventilation has been with us for over 100 years but that the acceptance of the technology has been very variable. The Draeger Pulmotor did not arouse the interest it deserved from the medical profession and it took several decades before the development and use of the modern portable gas—powered ventilator. Even today there is a wide variation in the use of portable ventilators in the prehospital setting, depending on training and culture. These devices are more widely used in Europe than the USA for example. This is probably due to the belief that the self reforming ‘Ambu’ bag offers a safer option for emergency ventilation than a PGPV in the hands of a non-expert. The reasons why this is not necessarily true are discussed further in Chap. 5.

At the time of preparing the second edition of this book the COVID19 pandemic has swept the world with more than 21 million reported cases and three quarters of a million deaths. Sadly, these figures are likely to rise substantially until an effective vaccine is found. The most severely affected cases require AV, sometimes for an extended period. Since the 1918–1919 influenza pandemic there has not been an event on this scale and nothing could have brought home more to the providers of medical care and the general public the importance of AV. Despite repeated warnings from medical voices over recent years of the need for mass ventilation many countries have found themselves unprepared and without the necessary stockpiles of devices. The COVID19 outbreak has led to a world shortage of mechanical ventilators and importantly the connecting circuits and airway devices that go with them, a situation that currently remains unresolved in many countries. This brief survey of the history of AV therefore ends at a time there has never been a greater need for an understanding of the subject and that provisions will be made in providing equipment and training to better face any future disaster of the scale currently seen.

## 1.11 Conclusions

1. This brief review of AV from earliest times until the early 1980s has covered many discoveries and re-discoveries based upon the increasingly recognised need to provide respiratory support to avoid death from hypoxia.

2. The key modern turning point which influenced respiratory care was undoubtedly the 1952 polio epidemic where, for probably the first time it was realised that death was from hypoxia rather than from the effects of the underlying disease process. Since that time AV, along with airway support has been fundamental to the practice of medical care both inside and particularly outside the hospital.
3. AV is a basic part of the ABCDE (airway, breathing, circulation, disability exposure) approach to trauma management, particularly for head injury. In neonatal practice the provision of AV has improved survival from premature birth to a level that would have been inconceivable 100 years ago. In the area of basic life support for cardiac arrest, simple positive pressure ventilation provided by exhaled air is now an established part of first aid training.
4. However, along with this expansion of the use of AV outside the hospital there has been a divergence in the equipment and training required to operate the increasingly complex ventilators found in the modern ICU and the more straightforward devices available for emergency care.
5. The management of AV in hospital has become increasingly the realm of specialists, both in nursing and the sub-speciality of the respiratory therapist in the United States. Intensive care medicine itself has broken away from its roots in anaesthesia and is now a separate speciality with physicians who are able to grasp the complexities of modern ventilators and apply their use scientifically in the ICU. With the development of such specialities has come an increasing awareness that early ventilation may have been damaging the lungs by the application of too high a pressure and excessive tidal volumes. This led to a major change in hospital ventilation policy about 15 years ago to provide ventilation that was far more adapted to the delicate nature of the lung.
6. Ventilation in the prehospital setting has yet to follow the path taken by practitioners in the hospital ICU and there is still a gulf between hospital and prehospital providers of AV. In the following chapters the theory and practice of AV is presented for the non-specialist. These will include medical, paramedical and nursing staff who may need to provide AV in emergency and for situations such as mass epidemics.
7. The discussion will concentrate on the essential anatomy and physiology of respiration and will cover largely equipment that is likely to be used by the non-specialist. Although the main emphasis of the text will be on AV for emergencies and the transport of ventilator—dependent patients this new edition will try to explain to non-specialists the approaches to AV taken by intensive care staff and the equipment they use.
8. There are many excellent detailed texts that cover AV for specialists and the complex equipment used and the interested reader requiring more information is referred to these, some of which are listed in the suggestions for further reading.
9. The COVID 19 pandemic has caused mass world-wide and highlighted the importance of AV and how to provide it. There has never been a greater need for a wider understanding of the subject and the challenges it presents.

## Suggestions for Further Reading

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# Chapter 2

## The Structure of the Airways and Lungs

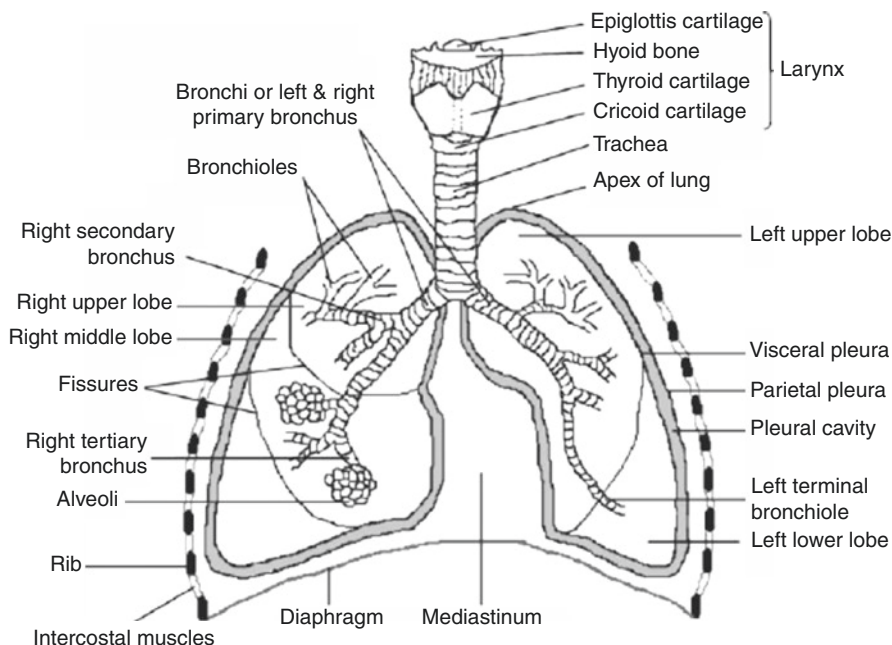


### 2.1 Introduction

A basic understanding of how the respiratory system is constructed and functions is essential to effective artificial ventilation. With the development of our knowledge of the anatomy and physiology of the lungs over the past 200 years the subject is complex and there are many specialized texts. The objective of this chapter is to provide a basic overview of the structure of the respiratory system and how the anatomy relates to the practical application of artificial ventilation. Key points are presented where these have a practical bearing on the clinical management of respiratory failure and artificial ventilation, particularly in relation to emergency ventilation. The essential function of the respiratory system, considered in the next chapter is the exchange of oxygen and carbon dioxide with the blood.

### 2.2 The Respiratory Tree: An Overview

The pathway from the mouth to the furthest recesses of the lungs is known as the respiratory pathway or tree and is divided between upper and lower airways. The upper airways comprises the nasopharynx, larynx the trachea and the main bronchi which allow the flow of gases in and out of the lungs. In addition these structures have an important function in warming and humidifying air to prevent contamination of the lower airways. The lower airway (which starts below the larynx) is made up of the trachea, the right and left main bronchi which then divide into 23 divisions to take inspired air to all parts of the lungs. After 23 divisions the airway continues to the terminal bronchi. Beyond the terminal bronchi are the acini where gas exchange with the blood begins. The acini are defined as the respiratory



**Fig. 2.1** The general anatomy of the upper and lower airways (Reproduced with permission, Neupac ventilation. Luton: Smiths Medical International) larynx cricoid cartilage

bronchioles, alveolar ducts and alveolar sacs. The alveoli are the final structures in the respiratory pathway where exchange of oxygen and carbon dioxide with the blood takes place. The terminal bronchi and acinus are sometimes called the respiratory lobule. Figure 2.1 shows the overall anatomy of the upper and lower airways.

### 2.2.1 Dead Space

All the upper and lower connecting structures of the airway provide dead space. This is the proportion of gas that is taken in during a single respiration which does not take part in exchange with the blood. Dead space is an important concept in practical artificial ventilation in deciding how much gas should be pumped into the patient during inspiration (see Chap. 6). Dead space is different between children and adults. In the adult it is about 150 ml. This is not related to weight. It is important to note that all the airways above the level of the terminal bronchi contribute to the anatomical dead space of the lungs. Only the acini are involved in gas transfer and do not therefore contribute to the dead space. In neonates and infants dead space varies with age as discussed in Chap. 9.

## 2.3 Passive and Active Airways

Another important difference between upper and lower airway structures is that the former are passive structures and do not respond to pharmacological actions or to immune related changes such as constriction of the bronchi. The upper airway can become blocked in emergency with secretions and vomitus and clearing the upper airway is an essential first stage in the provision of artificial ventilation in an emergency. The lower airways contain smooth muscle cells which are controlled by the autonomic nervous system. This means that they can constrict in reaction to immune challenges, as in bronchial asthma and can be affected by drugs such as adrenaline and salbutamol which can counteract such constriction.

The lower airways provide the lung with several defence mechanisms to protect the all-important but fragile alveoli. These include the cough reflex and the production and clearance of mucus. Mucus is produced by the epithelial cells and is swept up the airway by special cells with projections called cilia (Fig. 2.2). The mucus then moves to the upper airway and is coughed out. If the epithelium of the lower airways is damaged or the cilia do not function correctly the defence mechanisms malfunction and foreign substances can enter the respiratory lobules and cause lung inflammation.

## 2.4 The Alveoli

The terminal bronchi or bronchioles lead to the alveoli where exchange of oxygen and carbon dioxide with the blood takes place. The alveoli are delicate thin walled structures which are easily damaged. The key points about the anatomy of the alveoli is that gas exchange by simple diffusion can take place rapidly with the fine blood vessels of the lung tissue, known as pulmonary capillaries. The spaces

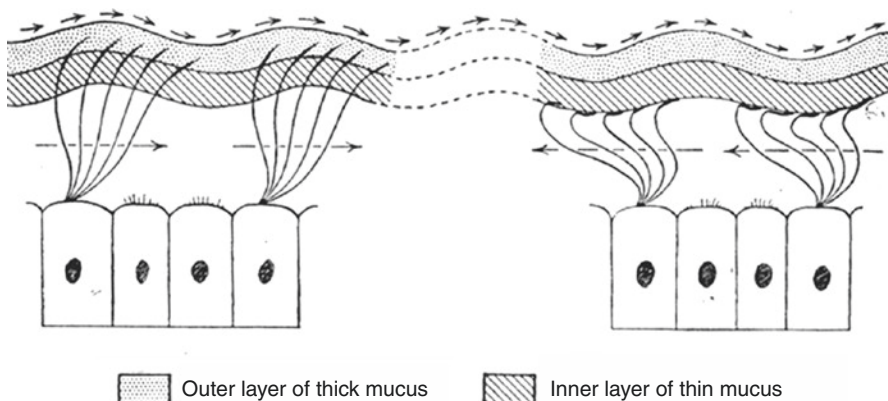


Fig. 2.2 Ciliated epithelium in the bronchi

between the alveoli, known as interstitial spaces play an important role in keeping the lung clear of fluid. If the alveolar sacs fill with fluid, which can happen in heart failure or following exposure to irritant toxic gases, gas exchange is disrupted and one form of respiratory failure follows. The delicate structure of the alveoli makes it a vulnerable site for attack by bacteria and viruses as well as inhaled toxic material. Also there is a fine balance between the clearing of the interstitial spaces and the operation of the heart.

## **2.5 Key Points of Respiratory Anatomy Relevant to Artificial Ventilation**

In this section we consider key points about the main anatomical structures making up the respiratory tree and how they can affect the practical management of the airway and artificial ventilation.

### **2.5.1 *The Nose***

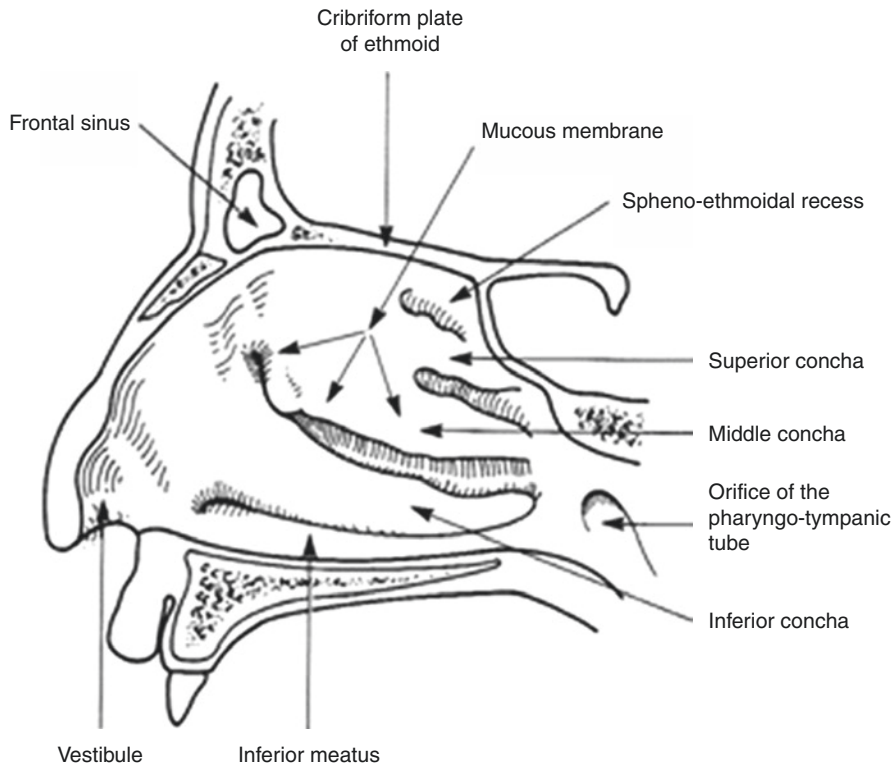
The nose extends from the nostrils anteriorly to the nasopharynx where it joins the extension of the mouth (Fig. 2.3). There are thus two main passages for external air to reach the lungs. In normal breathing at rest the mouth is closed and air passes through the nose. The nasal cavity is made up of bone and cartilage. It is divided into two halves by the nasal septum and is flanked by convoluted projections of bone called turbinates. The function of the nose is to provide passage of air and its filtration, humidification and warming. It also has a major function in detection of odour.

#### **2.5.1.1 Key Points Relative to Airway and Ventilation Management**

1. In emergency, the nasal cavity provides a valuable route to ensure the airway by nasal intubation (Fig. 2.4). However, insertion of a nasal airway or endotracheal tube may be difficult due to a deviation of the nasal septum and enlarged turbinates.
2. The nose has a rich blood supply and haemorrhage is possible from all points following trauma or intubation attempts.

### **2.5.2 *The Mouth (Oral Cavity)***

The oral cavity starts at the lips and leads back to the pharynx, which starts at the tonsils and the palatoglossal folds. The mouth contains the teeth and the anterior two thirds of the tongue. The tongue continues backwards into the pharynx and is



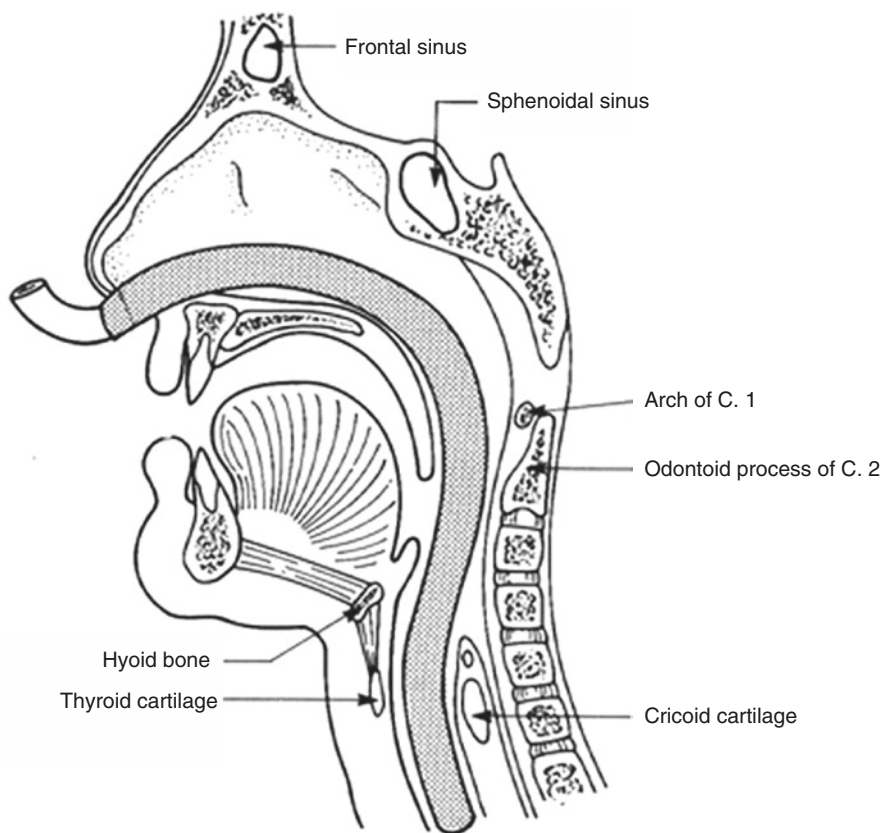
**Fig. 2.3** The general anatomy of the nose and mouth (Reproduced with permission from Aitkenhead and Smith. *Textbook of anaesthesia*. 2nd ed. London: Churchill Livingstone (Elsevier); 1990)

connected to the epiglottis by folds of mucosal tissue called the vallecular fossae. The main bony support for the mouth is the hard palate above and the mandible (jaw bone). This moves up and down in connection with the skull through the temporomandibular joint whose action determines how wide the mouth can be opened.

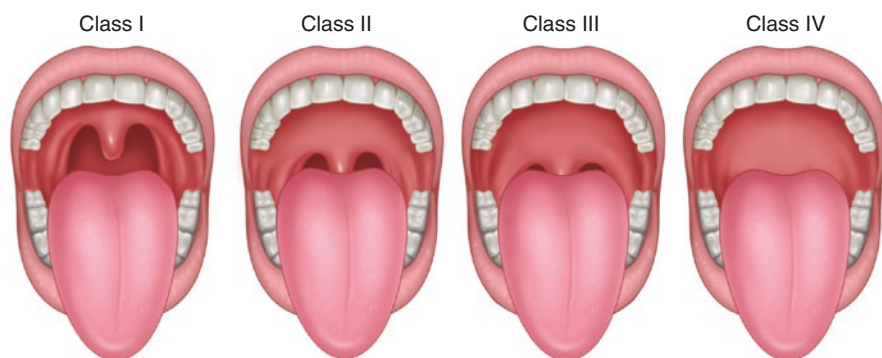
### 2.5.2.1 Key Points

1. The mouth is the main point of access in establishing an airway for artificial ventilation. In unconsciousness the bulky tongue is relaxed and blocks the passage of air in a person lying on his back. The essential primary airway measures (placing the person on his side, head tilt, chin lift and jaw thrust as discussed in Chap. 5) are all designed to overcome this problem.
2. Mouth opening can be very variable. In assessment before giving a general anaesthetic it is assessed by asking the patient to open his mouth and to see what is visible (the Mallampati classification—Fig. 2.5). However, this system is not





**Fig. 2.4** The nasopharynx (Reproduced with permission from Aitkenhead and Smith. *Textbook of anaesthesia*. 2nd ed. London: Churchill Livingstone (Elsevier); 1990)



**Fig. 2.5** The Mallampati classification of the visibility of the vocal cords during intubation

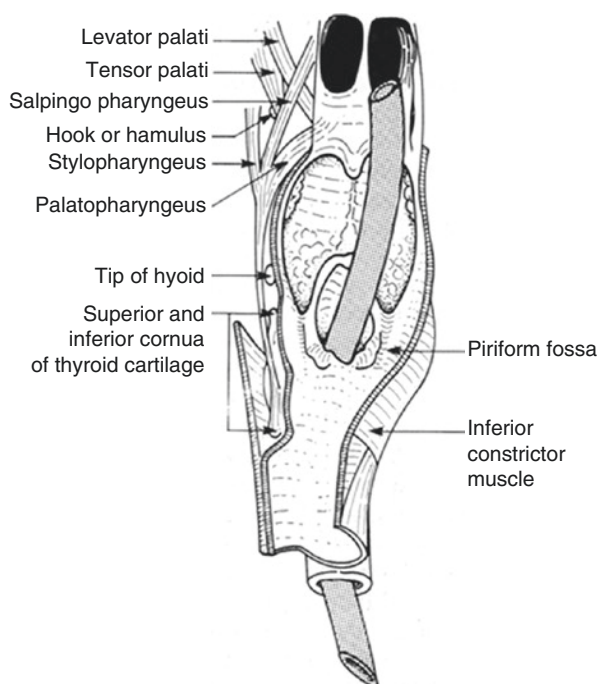
usually applicable to emergency management of the airway. A rapid inspection of the patient will give useful indicators to difficulties in intubation such as protruding teeth and a receding jaw.

3. The bulk of the anterior tongue can be depressed in the unconscious patient to allow passage of air using the Guedel airway. The shape of the mouth and tongue means that the device must be inserted with the curve facing up before rotation to control the tongue.

### 2.5.3 The Pharynx

The pharynx, (Fig. 2.6) situated at the back of the throat is a U-shaped structure with three distinct sections the nasopharynx, the oropharynx and the hypopharynx. The functions of the pharynx are (1) to act as an air passage (2) provide a common pathway for air and food and (3) to provide a passage to the oesophagus. The hypopharynx is important for airway management. The larynx bulges into the hypopharynx creating small recesses on either side known as piriform recesses.

**Fig. 2.6** The pharynx  
(Reproduced with permission from Aitkenhead and Smith. *Textbook of anaesthesia*. 2nd ed. London: Churchill Livingstone (Elsevier); 1990)



### 2.5.3.1 Key Points

1. The soft tissues around the larynx provide a bed for the seating of the laryngeal mask airway (a major alternative to endotracheal intubation—see Chap. 5)
2. Foreign bodies such as fish bones often lodge in the pyriform recesses
3. During intubation the tip of the endotracheal tube may slip into the pyriform recesses

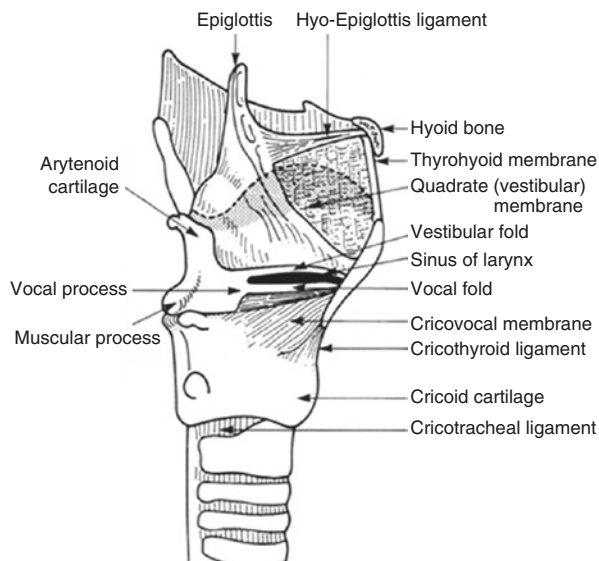
## 2.5.4 The Larynx

The larynx (Fig. 2.7) is an important structure in the upper airway which essentially acts as a valve protecting the airways from foreign substances such as food and secretions. It also produces vocal sounds. It is formed of cartilages, ligaments, membranes and small muscles. The structure is complex but essentially the larynx consists of three unpaired cartilages, the epiglottis, and the thyroid and cricoid cartilages and three paired cartilages, the arytenoid, cuneiform and corniculate. The unpaired cartilages make up the overall structure of the larynx while the paired cartilages control the essential function of the larynx through the vocal folds.

### 2.5.4.1 Key Points

1. The epiglottis acts to protect the upper airways from contamination the alimentary tract

**Fig. 2.7** The larynx  
(Reproduced with permission from Aitkenhead and Smith. *Textbook of anaesthesia*. 2nd ed. London: Churchill Livingstone (Elsevier); 1990)



2. Below the thyroid cartilage lies the cricoid cartilage. This is the only complete cartilage ring in the larynx. Between the thyroid and cricoid cartilages is the cricothyroid membrane. This provides an important point of access to the upper airway in emergency when the larynx is blocked
3. Endotracheal intubation involves passing a tube through the larynx via the vocal cords. These delicate structures may be damaged during insertion of the tube
4. The cricoid cartilage continues behind the thyroid cartilage to form the true vocal cords which are attached to the arytenoid cartilages. These are attached to the side of the thyroid by the aryepiglottic ligaments. They also articulate against the cricoid cartilage below. The tension of the vocal cords and the patency of the larynx and epiglottis is controlled by external and internal groups of muscles. The external group controls the position and movement of the larynx as a whole while the internal muscles control the delicate movements of the larynx that control the opening of the glottis. An exception is the cricothyroid muscle which lies outside the larynx and which serves to provide tension to the vocal cords via the arytenoid cartilages
5. The vocal cords may become tightly closed in a condition known as laryngeal spasm. This is as a result of stimulating the underside of the epiglottis. Touching the upper side of the epiglottis (the usual situation during endotracheal intubation) does not produce spasm since the nerve supply here is different, coming from the ninth cranial nerve (the glossopharyngeal nerve) and not the tenth cranial nerve (the vagus) which also provides the delicate muscle control of the larynx
6. The practical implications of these key points about the larynx are discussed in Chap. 5

### 2.5.5 The Trachea

The trachea (Fig. 2.1) is a tubular cartilaginous structure which begins at the lower part of the cricoid cartilage at the level of the sixth cervical vertebra. It consists of 16–20 C—shaped cartilaginous partial rings, joined by a membrane behind. The adult trachea is about 12 mm in diameter and 9–15 cm in length. The trachea divides into the right and left main bronchi at a point known as the carina. This is situated at the level of the fourth and fifth thoracic vertebrae.

#### 2.5.5.1 Key Points

1. The trachea can be opened surgically by tracheostomy to provide emergency access to the airway. However, this is a surgical procedure and is not appropriate during emergency for non-specialists. Instead, access through the cricothyroid membrane should be used (Fig. 2.7)

2. The division of the trachea into the right and left main bronchi means that an endotracheal tube which is too long will enter the right main bronchus, since this is more in line with the direction of the trachea. To avoid this, adult tubes should be cut to a length of about 23 cm which is the average distance from the lips to the mid-point of the trachea

### ***2.5.6 The Upper and Lower Bronchi***

After the division of the trachea at the carina into the right and left main bronchi there is a gradual branching of the bronchi down to the level of the respiratory lobule and the alveoli. In all, there are 23 generations or levels of airway between the trachea and the alveolar sacs. The key anatomical features of these airways are as follows:

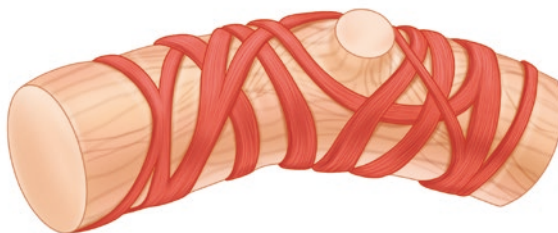
#### **2.5.6.1 The Main Bronchi (1st to 4th Generations)**

These structures have a firm cartilage support which ensures their rigidity in the negative pressure surroundings of the chest cavity. There is a characteristic U shape at the upper levels but this is replaced by irregular shaped cartilage bands at lower levels. After this the form of the bronchi begins to be supported by smooth muscle bands, arranged in a geodesic formation (Fig. 2.8).

#### **2.5.6.2 The Small Bronchi (5th to 11th Generations)**

During this evolution, the size of the bronchi diminishes from 3.5 to 1 mm. But an important point is that since the number of bronchi doubles with each generation the *total* cross sectional area of the bronchi increases considerably with each generation. At the level of the 11th generation the cross-sectional area is about seven times that of the lobar bronchi. This is important for ventilation of the lungs because the cross-sectional area determines the resistance of the airway to the flow of gas within

**Fig. 2.8** The geodesic structure of bronchiolar muscle



it. In practice, during artificial ventilation this means that the main contribution to airway resistance comes from the larger rather than the smaller bronchi.

### 2.5.6.3 Bronchioles (12th to 16th Generation)

At the level of the 11th generation of bronchi there is a major change in the structure of the airway in that cartilage disappears from the walls and the structural rigidity is no longer the main factor in determining airway patency (Box 2.1). From this level the airways are held open by the elastic recoil of the lung tissue (parenchyma) which hold the bronchi open rather like the guy ropes of a tent. This means that the calibre of the airways below the 11th generation is mainly influenced by lung volume and is less influenced by intrathoracic pressure than in the case of the upper bronchi.

The nervous control of the bronchioles is very important for artificial ventilation since it is a major determinant of airway resistance and gas flow. Bronchioles receive the nerve supply from the involuntary (autonomic) nervous system and have both a sympathetic and parasympathetic supply. Discussion of these two sections of the autonomic nervous system is beyond the scope of this book but the reader will find details in basic physiological texts. Essentially however the key practical points for ventilation are that the sympathetic system acts through nor adrenaline as a transmitter of nerve impulses to the smooth muscle fibres while the parasympathetic system acts through acetyl choline. This explains why drugs acting on these two systems can have such immediate effects in emergency. Importantly, reversing bronchoconstriction where the bronchi suddenly contract as in asthma or allergic reaction and also in poisoning by anticholinesterases such as nerve gases can be achieved (1) by administering drugs such as adrenaline, ephedrine and salbutamol

#### **Box 2.1 The Structure of the Bronchi and Resistance to Gas Flow**

Beyond the 11th generation of bronchioles the number increases more than their calibre diminishes. This means that the total cross sectional area increases up to more than 90 times that of the main bronchi. This explains why the resistance to gas flow at the level of the smaller airways (<2 mm diameter) is only one tenth of the total airway resistance. But it also explains why airway resistance increases so dramatically when the bronchioles, surrounded by their smooth muscle constrict suddenly, as in the case of an asthmatic attack. Bronchioles have a geodesic arrangement of bronchial muscle bands (a geodesic is the shortest distance between two points on a spherical surface) which is the best way of ensuring the patency of the tubular airway structure without the muscle bands tending to slip along the surface.

Moving further down the bronchial tree, the muscle around the bronchioles becomes thinner but the relative thickness in relation to the wall dimensions increases. In a bronchiole with 1 mm diameter the muscle bands are five times as strong as in a bronchiole of 11 mm diameter.

which imitate the action of noradrenaline (known as sympathomimetic drugs) or (2) blocking the action of the acetyl choline in the parasympathetic supply as in the case of the use of atropine in nerve agent poisoning.

#### 2.5.6.4 Respiratory Bronchioles (17th to 19th Generation)

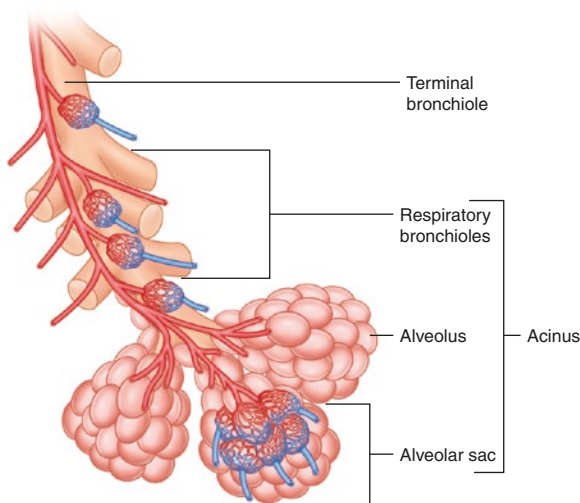
Up to the level of the 16th generation the function of the airway passages is to ensure the flow of air and its humidification. Beyond this point the bronchioles increasingly take part in the exchange of gas with the blood in the pulmonary capillaries until, at the level of the alveolar ducts the whole surface of the airway is devoted to gas exchange. As noted above, the fragile respiratory bronchioles are embedded in the lung structure (parenchyma) and are kept open by elastic traction. Beyond the 19th generation of bronchioles, the end of the respiratory tract passes into the respiratory lobules which are the functional units of gas exchange in the lungs (Fig. 2.9). Each lobule contains about 2000 alveoli. There are about 130,000 primary respiratory lobules.

Finally the respiratory pathway develops into generations 20–22 which are passages leading to the alveoli (the alveolar ducts) and finally to generation 23 which is the alveolar sac. The final division constitute the acinus (Fig. 2.9).

#### 2.5.6.5 The Alveoli

Knowledge of the basic structure and function of the alveolus is essential for the understanding of how artificial ventilation works in overcoming respiratory failure.

**Fig. 2.9** The respiratory lobule



There are about 15–20 alveoli in each alveolar sac. These are about 0.2 mm in diameter but this figure is affected by the state of inflation of the lung and the weight of lung tissue above when standing upright which compresses the lower lung tissue. In the alveoli, the air and blood are separated by a thin layer of tissue about 1–2  $\mu$  thick. This is made up of four layers which are important for the function of the alveoli following the failure of gas exchange in type 1 respiratory failure (see Chap. 4).

The layers are as follows:

1. The alveolar lining fluid or surfactant

This important substance reduces the surface tension inside the alveolus which would otherwise tend to collapse. Absence of surfactant in premature babies causes the serious condition of respiratory distress syndrome. Importantly, it is now known that prolonged exposure to high concentrations of oxygen may also lead to reduction of surfactant. This has brought about a major change in policy in recent years for long term artificial ventilation which is discussed later.

2. The alveolar epithelium

This contains two important cells known as type 1 and type 2 cells (Fig. 2.10) Type 1 cells are the real alveolar epithelium cells spreading to form the walls of the alveoli. Type 2 cells are different in shape (they are cuboidal) and are involved in the production of surfactant.

3. The interstitial layer

This is a very thin layer between the alveolar epithelium and the capillary endothelium.

4. The capillary endothelium

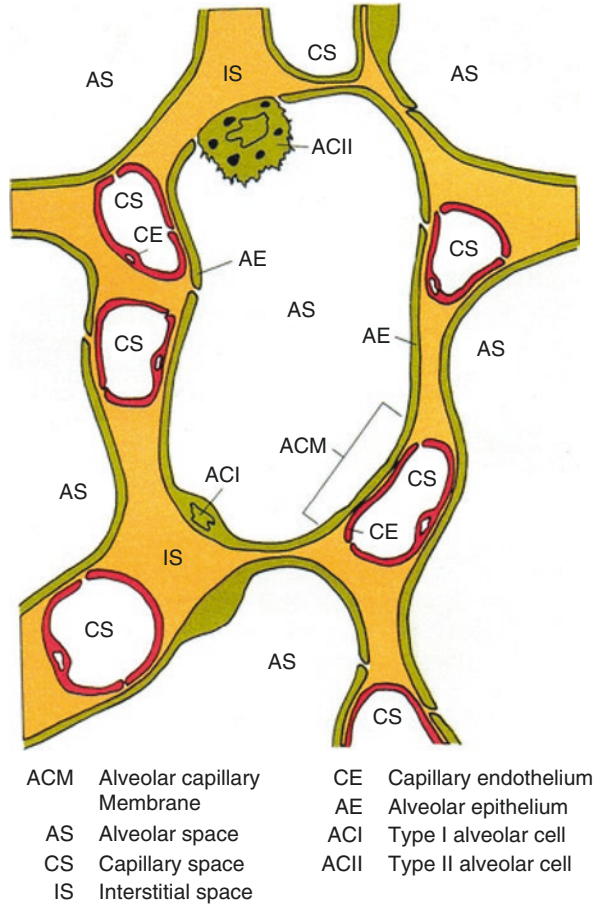
This forms the walls of the pulmonary capillary and allows diffusion of oxygen and carbon dioxide to and from the blood. The dividing walls (or septa) between the alveoli consist of mirror images of layers 1–3 above with the capillary network in between. This is dense in some places but on average is in contact with about 75% of the total alveolar surface. The space between the adjacent alveolar lining layers and capillaries is known as the interstitial space and is filled with elastic and collagen fibres, smooth muscle and nerves.

The properties of the structures of the alveoli and pulmonary capillaries give rise to a delicate balance of maintaining the levels of fluid in the interstitial spaces. This is dependent upon the pressure of blood in the capillaries and the osmotic properties of the blood and tissues. The overall relationship is given by the Starling Equation which is discussed in Chap. 3.

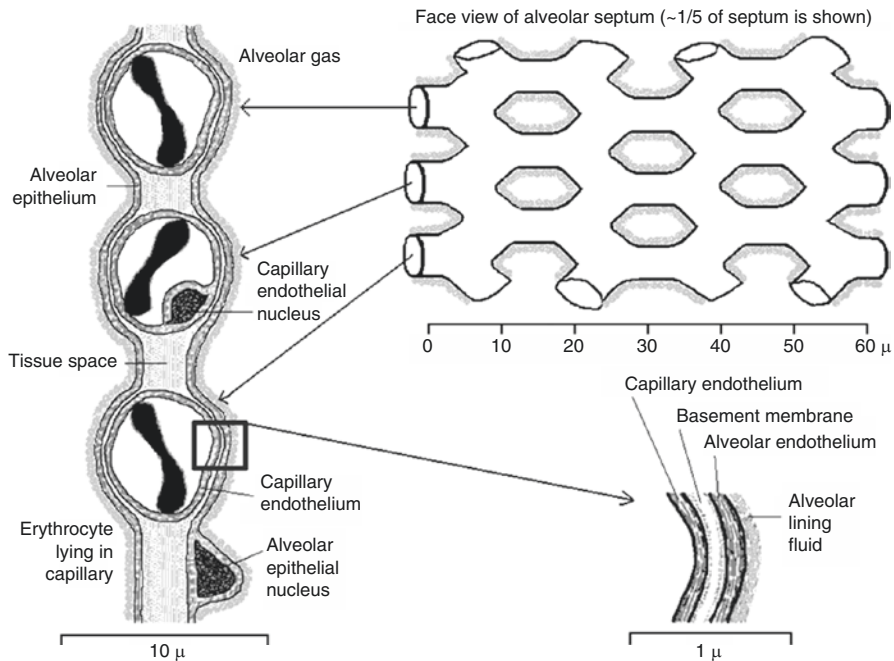
The structure and patency of individual alveoli are determined partly by gravity and partly by the elastic forces of the lung acting against the negative inspirational pressure which leads to normal air entry and also by reduction of surface tension in each sac due to a lipoprotein film, (surfactant). This forms a thin covering over the alveolar epithelium. The alveoli have a direct and intimate contact with the pulmonary capillaries. The distribution is however not symmetrical there being 'thin' and



**Fig. 2.10** The structure of the alveoli (Reproduced with permission, Smiths Medical International, Luton UK)



‘thick’ sides. The thin side is only 0.4 micron thick and contains alveolar epithelium and capillary endothelium separated only by the cellular and basement membranes. The ‘thick’ side measures about 1.2 microns and has an interstitial space containing elastin, collagen and possibly nerve fibres. This side takes less part in gaseous exchange than the other side and its role is largely structural, providing a support for the alveolus against potential collapse. The size of the pulmonary capillaries allows the passage of only one haemoglobin containing erythrocyte at a time. These cells are concave and have the maximum surface area for a given diameter optimising oxygen exchange. Figure 2.11 shows one enlarged view of the wall of an alveolus (a) and a cross sectional view of the relationship with the pulmonary capillaries.



**Fig. 2.11** The alveolar septum (Reproduced with permission, Smiths Medical International, Luton, UK)

## 2.6 Conclusions

1. A basic understanding of the structures of the respiratory pathway is important for artificial ventilation. The respiratory pathway is called the respiratory airway or tree
2. Airways can be divided into upper and lower. The upper and most of the lower airways are involved in the humidification of air during breathing and its conduction to the alveoli where oxygen exchange with the blood takes place
3. Both the upper and lower airways create dead space. This is the proportion of the air during each breath which is not involved in gas transfer
4. There are important sites in the anatomy of the respiratory pathway which are relevant to keeping it open and for the application of artificial ventilation
  - The nasopharynx, which can become blocked as a result of the patient's position when unconscious
  - The larynx, which acts as a protective entry valve to the trachea and bronchi
  - The cricothyroid membrane and trachea which can provide sites for emergency access to artificial ventilation when the nasopharynx and larynx are blocked

- The upper and lower bronchi which divide into 23 generations. These have a total cross sectional area greater than that of the trachea and main bronchi and during normal breathing contribute less to airway resistance. The diameter of the lower bronchi can be reduced greatly by contraction of smooth muscle in the walls as in an asthmatic attack. This increases airway resistance considerably
- Beyond the 19th generation of small bronchi the respiratory tract passes into the respiratory lobules which are the functional units of gas exchange in the lungs

## **Suggestions for Further Reading**

Lumb AB. Nunn's respiratory physiology. 7th ed. London: Churchill Livinstone; 2010.

West JB. Respiratory physiology: the essentials. 7th ed. Philadelphia, PA: Lippincott, Williams and Wilkins; 2005.

## Chapter 3

# How the Lungs Work: Mechanics and Gas Exchange with the Blood



### 3.1 Introduction and Definitions

This chapter deals with the way the lungs work, how they interact with the blood flowing through them and with the way oxygen is used by the cells of the body. The flow of oxygen and carbon dioxide in and out of the body is assured by breathing. This term is often used interchangeably with 'respiration.' However, breathing may be more accurately defined as the movement of the chest and diaphragm (the thorax) to cause the flow of gases in and out of the lungs. Respiration is strictly defined as (1) external respiration, where gas is exchanged with the blood in the lungs and (2) internal respiration, where oxygen is metabolized in the cells of the body. The term 'ventilation' is used to describe the physical flow of gases in and out of the lungs during breathing. Thus, artificial ventilation is the replacement of this natural process by mechanical means. These definitions are summarized in Box 3.1.

#### Box 3.1 Basic Definitions

*Breathing* is the mechanical process by which air is sucked into the lungs from the outside. Breathing is an automatic physiological process which can be overridden by voluntary effort. The entry of air into the lungs follows the creation of a negative pressure inside the thorax by the downward contraction of the diaphragm and the intercostal muscles of the chest wall.

*Ventilation* is strictly a measure of the flow of air that passes in and out of the lungs during normal breathing and in artificial ventilation.

*Respiration* is the process of transfer of air to the blood (*external respiration*) and use of oxygen in the cells to produce energy (*internal respiration*).

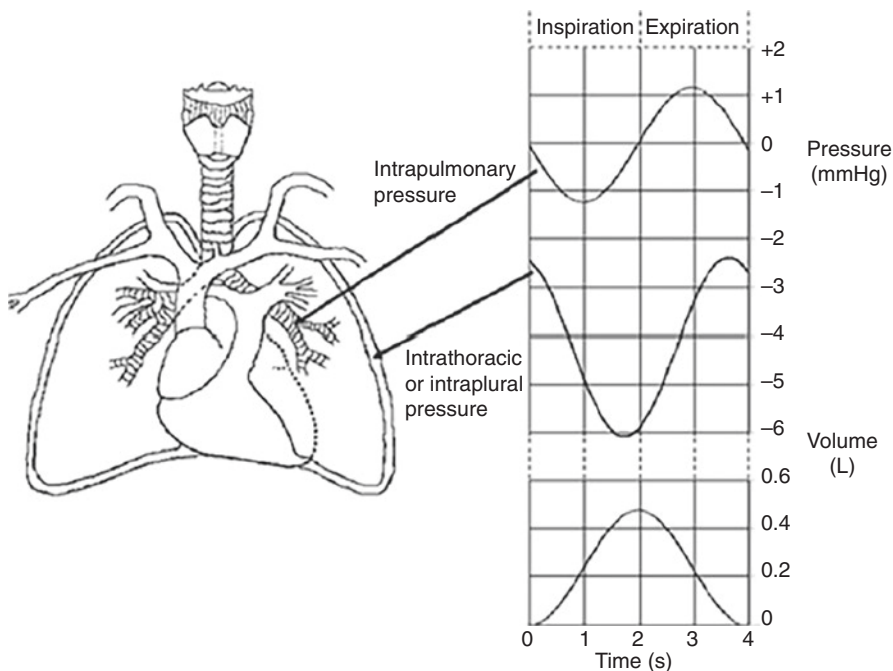
## 3.2 The Mechanics of Breathing

### 3.2.1 The Normal Ventilation of the Lungs

Breathing creates a partial vacuum inside the chest cavity which draws air into the lungs from outside. There is thus a small negative pressure inside membranes sealing the chest, known as the pleural cavity. The relation between this negative pressure and the flow of air into the lungs is shown in Fig. 3.1. This ideal situation is affected by the properties of the chest wall and the lung tissue or lung mechanics. These properties are important for the understanding of the artificial ventilation of lungs using positive pressure.

### 3.2.2 Airway Resistance and Lung Compliance

The anatomical structures of the airways and lungs discussed in Chap. 2 determine two important features which are fundamental to the understanding of artificial ventilation of the lungs. These are airway resistance and lung compliance.



**Fig. 3.1** Negative pressure in the chest cavity and airflow into the lungs (Reproduced with permission, Pneupac Ventilation, Smiths Medical International, Luton, United Kingdom)

### 3.2.2.1 Airway Resistance

When gas passes into the lungs it must overcome a degree of obstruction to its passage which is determined by the diameter of the airway structure through which it is passing. Airway resistance is different between the upper and lower airways. Because of the limited number of airway passages, the resistance in the wider upper normally is proportionally greater than the lower airways, which although smaller in diameter are numerically greater. However, the resistance in the bronchioles is affected by many factors such as the muscle tone of the smooth muscle in the bronchiolar walls, the thickness of the mucous membrane and the consistency of the secreted mucous. These factors play an important role in the distribution of gas around the lungs. Any section of the lung where local resistance is increased will be less—ventilated than others. Airway resistance is defined as pressure difference (usually measured in cm H<sub>2</sub>O) divided by the rate of flow of gas in litres/sec. The normal value in adults is usually 5 cm H<sub>2</sub>O/L/s. In conditions such as asthma the value may rise considerably up to between 20 and 50 cm H<sub>2</sub>O/L/s. This is an important consideration in the function of artificial mechanical ventilators (see Chap. 7).

### 3.2.2.2 Compliance

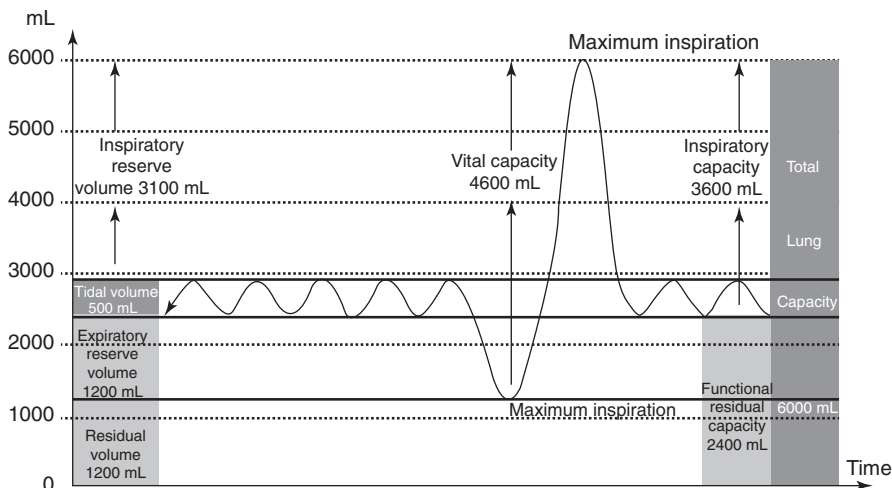
Compliance of the lungs is defined as the degree to which they can be stretched when a given pressure is applied. It is expressed as  $C = V/P$ . The units are usually litres per cm H<sub>2</sub>O. Overall compliance, which is a measure of how the chest expands during normal and artificial ventilation is made up of the compliance of the thorax which surrounds the lungs and the compliance of the lungs tissue itself. The relationship is

$$\text{Overall compliance} = 1 / C_{\text{lung}} + 1 / C_{\text{Thorax}}$$

Normal overall compliance is 0.1 L/cm H<sub>2</sub>O. This falls in a number of conditions such as pneumonia and acute respiratory distress syndrome. The water content of the lungs is the most significant factor in emergency situations (when pulmonary oedema may be present) and thus it is the lung tissue compliance which determines principally the observed changes which affect ventilation. Changes in compliance caused by changes in the elastic fibres of the lungs are usually only significant in the case of chronic lung disease.

### 3.2.3 Lung Volumes and Capacities

During normal breathing, a set volume of air passes in and out of the lungs. However this amount of air, known as the tidal volume is only part of the total volume of air in the lungs at any one time. In physiology the air in the lungs is measured by spirometry (a spirometer is a simple volume measuring device that can be connected to a spontaneously breathing patient). Figure 3.2 shows a normal spirogram which is made up of four volumes and four capacities (a capacity is the sum of several volumes).



**Fig. 3.2** The normal adult spirogram (Reproduced with permission, Pneupac Ventilation, Smiths Medical International, Luton, United Kingdom)

These are defined as follows:

**Tidal volume ( $V_t$ ):** the amount of air passing in and out of the lungs during normal breathing (500 ml).

**Inspiratory reserve volume (IRV):** the maximum extra inspiration that can be taken after a normal inspiration (3100 ml).

**Expiratory reserve volume (ERV):** the maximum additional forced expiration possible after a normal expiration (1200 ml).

**Residual lung volume (RV):** the gas remaining in the lung after a maximum voluntary expiration (1200).

These lung volumes can be combined to define lung capacities as follows:

**Inspiratory capacity ( $IRV + V_t$ )** (3600 ml).

**Vital capacity ( $IRV + V_t + ERV$ )** (4600 ml).

**Functional residual capacity ( $ERV + RV$ )** (2400 ml).

**Total lung capacity ( $IRV + V_t + ERV + RV$ )** (6000 ml).

In respiratory medicine the measurement of lung capacities gives useful information about the progress of lung diseases. In emergency and transport ventilation the most important of the parameters listed above are tidal volume and functional residual capacity.

### 3.2.3.1 Tidal Volume

Figure 3.2 shows that tidal volume ( $V_t$ ) is the volume of gas passing in and out of the lungs each time a breath is taken. It is also used in artificial ventilation to describe the volume of gas delivered to the patient (inspired tidal volume) and the volume during passive expiration (expired tidal volume). Tidal volume is dependent upon age and weight. For an adult the volume can be calculated as approximately 10 ml/

kg. For neonates and infants the relationship is approximately 15 ml/kg. In the normal adult only 350 ml of the tidal volume can take part in gas exchange in the alveoli since the dead space of the airways (the volume of air contained in the airways that does not take part in gas exchange with the blood) is about 150 ml. In artificial ventilation it is essential to ensure that the tidal volume does not fall below the value of the dead space, when there is no effective ventilation of the alveoli. It should be noted that in an intubated patient the anatomical dead space is reduced but there may be an associated extra dead space associated with the circuit connecting the ventilator to the endotracheal tube.

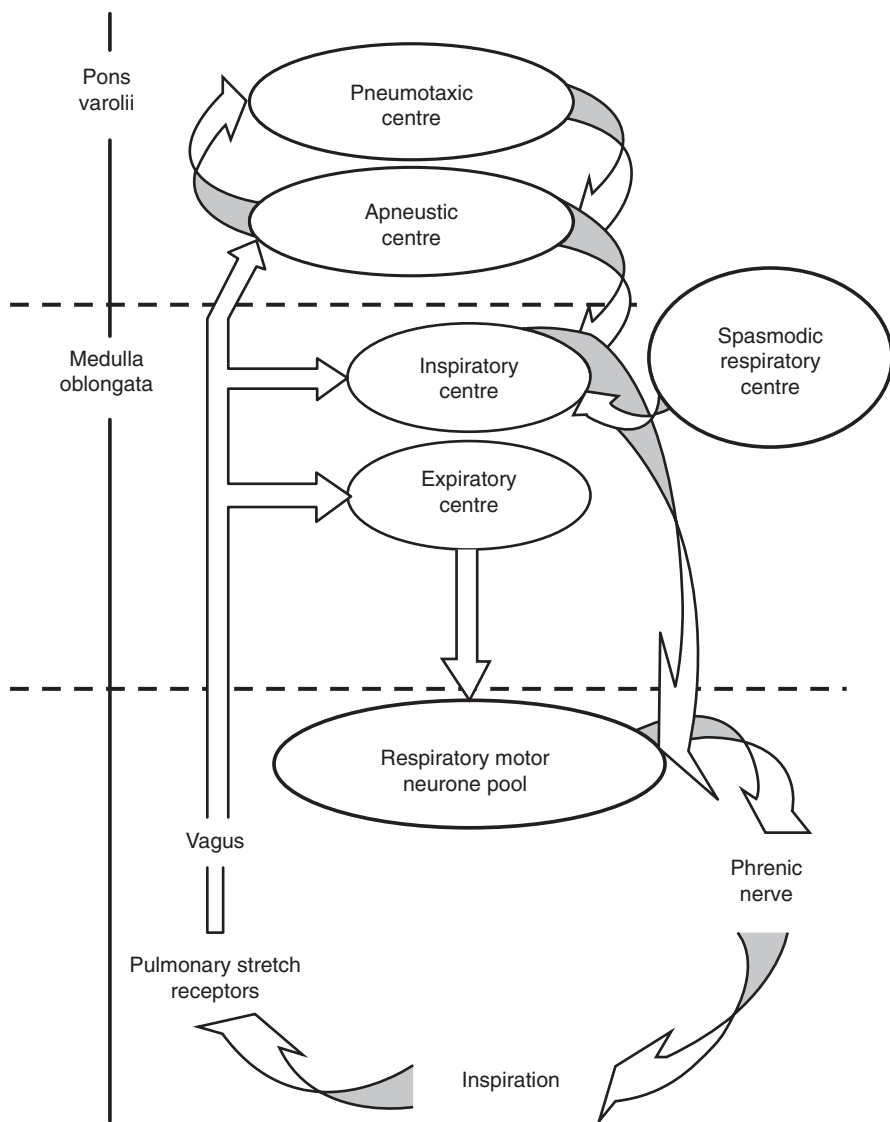
### 3.2.3.2 Functional Residual Capacity

The importance of functional residual capacity (FRC) is that it acts as a large buffer volume of inspired air to even out what would otherwise be large fluctuations in the concentration of oxygen in the alveoli. An adult has an FRC of about 2500 ml and about a fifth of this volume is exhaled during normal respiration and filled with fresh air. Atmospheric air contains oxygen at 21%. However, resting alveolar air contains 14% oxygen. Thus the FRC contains  $0.14 \times 2400 \text{ ml} = 350 \text{ ml}$  oxygen. If a tidal volume of 500 ml air containing 21% oxygen is inhaled an extra 105 ml of oxygen is added, making a total of 455 ml oxygen in the 3000 ml of total alveolar air. This changes the oxygen concentration in the FRC from 14 to 15.1%. Thus the large buffer volume provided by the FRC means that the fluctuations of the oxygen concentration in the alveolar air are only about 1% which changes the partial pressure by about 7 mm.

## 3.3 The Regulation of Breathing: An Overview

Breathing is an automatic process which can be overridden when oxygen demands from the body are increased. There are different physiological mechanisms which control both these situations. Breathing is regulated by a main control centre situated in the medulla oblongata at the lower part of the brain and by sensors situated in the blood vessels and lungs which send information to the control centre (Fig. 3.3). Automatic, normal ventilation takes place using a control (positive feedback) loop system. In this type of control system (which is also used in engineering) there is a sensor which monitors the current level of the variable to be regulated. The value transmitted is compared to a reference value and the control system responds according to whether the value should be increased or decreased. In the body this control loop system is provided by the medulla oblongata and by chemoreceptors, located in the walls of the carotid artery and the aorta which measure the levels of oxygen, carbon dioxide and the acidity of the blood. In normal automatic breathing impulses for the contraction of the respiratory muscles are sent out from the medulla oblongata (at the lower end of the brain) automatically from areas of nerve cells which control both inspiratory and expiratory activity. These centres are not under voluntary control.





**Fig. 3.3** The neural control of breathing (Reproduced with permission, Pneupac Ventilation, Smiths Medical International, Luton, United Kingdom)

### ***3.3.1 Inspiration and Expiration Control***

During intrinsic breathing (regular breathing at rest without any conscious effort) inspiration is triggered when the inspiratory centre neurones are electrically discharged. This discharge is relayed to the respiratory muscles along the motor nerve tracts and results in muscle contraction of the diaphragm and intercostal muscles of the chest wall. During expiration, the neurones of the inspiratory centre are inactive for 3s and recharge while those of the expiratory centre discharge and are then re-charged during inspiration. The inspiratory neurones then fire for 2s, stimulating the diaphragm and external intercostal muscles.

### ***3.3.2 The Neural Control of Breathing***

Normal breathing is controlled by a series of neural centres, shown in Fig. 3.3 situated in the floor of the fourth ventricle of the brain which provide a cyclical discharge to ensure regular active inspiration. If the higher centres are removed or damaged breathing continues in a rudimentary form in the form of gasping, from the apneustic centre (apneustic breathing). These control centres may be affected by trauma, raised intracranial pressure and toxic factors. Specifically the neural control centres are:

- The medullary centre, situated in the hind brain (in the floor of the fourth ventricle)
- The pneumotaxic and apneustic centres which are situated in the mid brain (the pons varolii)

The role of the medullary centre is to control the regular rhythm of breathing. It does this through sets of neurones which control both the inspiratory and expiratory timing. The inspiratory centre begins to fire after 3s of expiration during which it is inactive. While firing during 2s of inspiration it activates both the diaphragm and the external intercostals muscles producing the expansion of the thoracic cage. At the end of this time it ceases to fire and the inspiratory muscles relax while expiration occurs passively as described above. However higher levels of the brain can activate the expiratory centres voluntarily if a forced expiration is required. The medullary centre is autonomous and its activity continues even if the higher centres of the brain are put out of action.

The pneumotaxic centre in the pons is concerned with the co-ordination of inspiratory and expiratory activity, having an inhibitory effect on the inspiratory neurones. This means that it can regulate the degree of inspiration to prevent the lungs from over-distension. If the breathing rate rises the inspiratory time is shorter and the pneumotaxic centre is more active.

The apneustic centre in terms of evolution is the oldest and most basic of the respiratory control centres. Normally it provides stimulatory impulses to the inspiratory neurones and thus reduces expiration. It remains active when the pneumotaxic centre is not firing but is overridden by the higher centre. If the medullary and pneumotaxic centres are both non-functioning the apneustic centre provides a primitive, gasping form of basic respiration.

### ***3.3.3 Voluntary Override of Breathing***

All three of the neural centres interact to provide a co-ordinated system of breathing which is normally automatic. However higher cortical centres allow a voluntary override of the automatic control system for example in voluntary breath-holding when under water or when exposed to smoke. But the higher centres in the brain cannot override the control exerted by rising  $\text{CO}_2$  and acidity levels in the blood. When these reach a critical level the inspiratory centre is stimulated and involuntary breathing recommences. It is therefore not possible for a person to hold his or her breath to the point of death, although trained divers can remain apnoeic for very extended periods.

### ***3.3.4 Chemical Control of Breathing***

As mentioned above, breathing is also under the control of signals sent from special receptors (chemo-receptors) which detect changes in  $\text{CO}_2$  and oxygen in the blood. There are both central and peripheral receptors. The central receptors respond to pH and  $\text{CO}_2$  changes in the blood and cerebrospinal fluid around the brain while the peripheral receptors respond to pH,  $\text{PCO}_2$  and  $\text{PO}_2$  in the blood. The peripheral receptors are situated on the aortic arch and at the bifurcation of the carotid arteries.

Carbon dioxide produces an increase in acidity due to a breakdown of carbonic acid into hydrogen and bicarbonate ions. In addition  $\text{CO}_2$  can easily pass across membranes including those of the blood-brain barrier.

The peripheral chemoreceptors are stimulated (1) by increase in  $\text{CO}_2$  and (2) by a fall in arterial  $\text{pO}_2$  (from 100 to 50 mmHg (13.33 to 6.67 kPa)). When stimulation occurs signals are sent to the respiratory centres causing the rate and depth of breathing to increase. Hyperventilation follows and this causes the elimination of  $\text{CO}_2$  until the blood  $\text{CO}_2$  and pH again become normal.

Conversely, following hyperventilation, either voluntary or involuntary the blood  $\text{CO}_2$  levels are reduced and output from the chemo receptors stops, producing temporary apnoea.

## 3.4 Respiration and Gas Exchange with the Blood

### 3.4.1 Definitions

Respiration is defined as (1) external, which describes all the processes leading to the delivery of oxygen to the alveoli and (2) internal which concerns all processes relating to the transport of oxygen and carbon dioxide in the blood and the biochemical processes using oxygen and producing carbon dioxide in the cells of the body.

### 3.4.2 Gas Exchange in the Alveoli

The essential function of the alveoli is exchange of oxygen and carbon dioxide between the air contained in them ('alveolar air') with the blood. The average adult lung contains about 300 million alveoli which have a large surface area available for gas exchange with the pulmonary capillaries (80 square metres—about the size of a European football pitch). The alveoli are in contact with between 1000 and 4000 pulmonary capillaries. To reach the capillaries, oxygen must diffuse across the alveolar—capillary membrane, a distance of only 1 micrometre.

This membrane is a delicate structure which can be damaged by inhaling toxic gases and cause type 1 respiratory failure (see Chap. 4). Diffusion is a passive operation controlled by the concentrations of oxygen in the alveolus and capillary on either side of the membrane. Oxygen and carbon dioxide move from areas of high to low concentration. Thus oxygen in the alveoli which has a partial pressure (see Box 3.2) of 100 mm Hg diffuses into the venous blood returning to the lungs which

#### Box 3.2 Partial Pressure of Gases in a Mixture

Dalton's Law states that each gas in a mixture of gases exerts the same pressure as if no other gases were present. The pressure of a specific gas in a mixture is called the partial pressure. Air is a mixture of mainly oxygen, carbon dioxide, nitrogen and water vapour. At a standard temperature and pressure (20 °C and 760 mmHg) oxygen makes up 21% of the gas in air. Therefore in dry air the partial pressure of oxygen is:  $0.21 \times 760 = 160$  mmHg. Carbon dioxide is 0.04% and therefore its partial pressure is:  $p\text{CO}_2 = 0.0004 \times 760 = 0.3$  mmHg. Partial pressures of oxygen and carbon dioxide are conventionally used in physiology to express the amount of gases carried in the blood which is in contact with air in the lungs. For reasons given below this is not the same as the concentration of dissolved gas in the case of oxygen. Partial pressures are expressed as mm Hg (1 atmosphere pressure is equivalent to 760 mm) or as kilopascals (kPa) (one Pascal is a force of 1 kg applied over a surface of 1 square metre)  $1 \text{ kPa} = 7.5 \text{ mm Hg}$ .

has a partial pressure of 40 mm. In the pulmonary capillaries oxygen binds to haemoglobin in the blood red cells (erythrocytes). Haemoglobin greatly increases the capacity of blood to carry oxygen to the cells of the body—far greater than if oxygen were just dissolved in the plasma.

3.4.2.1 Composition of Gas in the Alveoli

At rest the main gases present in the lungs are nitrogen, oxygen and carbon dioxide. The lung is normally in contact with the atmosphere which exerts a pressure of 760 mmHg. However in the alveoli, water vapour is present and its partial pressure must be subtracted before calculating the partial pressures of oxygen and other gases present. Table 3.1 shows the partial pressures of gases in the alveoli as a percentage of the atmospheric pressure minus the partial pressure of water.

The partial pressures of the gases in the alveoli create pressure gradients when compared with the partial pressures of gases in the blood (that is, the pressure that would be measured above the blood if it were contained in a closed vessel).

Table 3.2 shows the partial pressure of gases at sea level in the pulmonary capillaries on the other side of the alveolar capillary membrane.

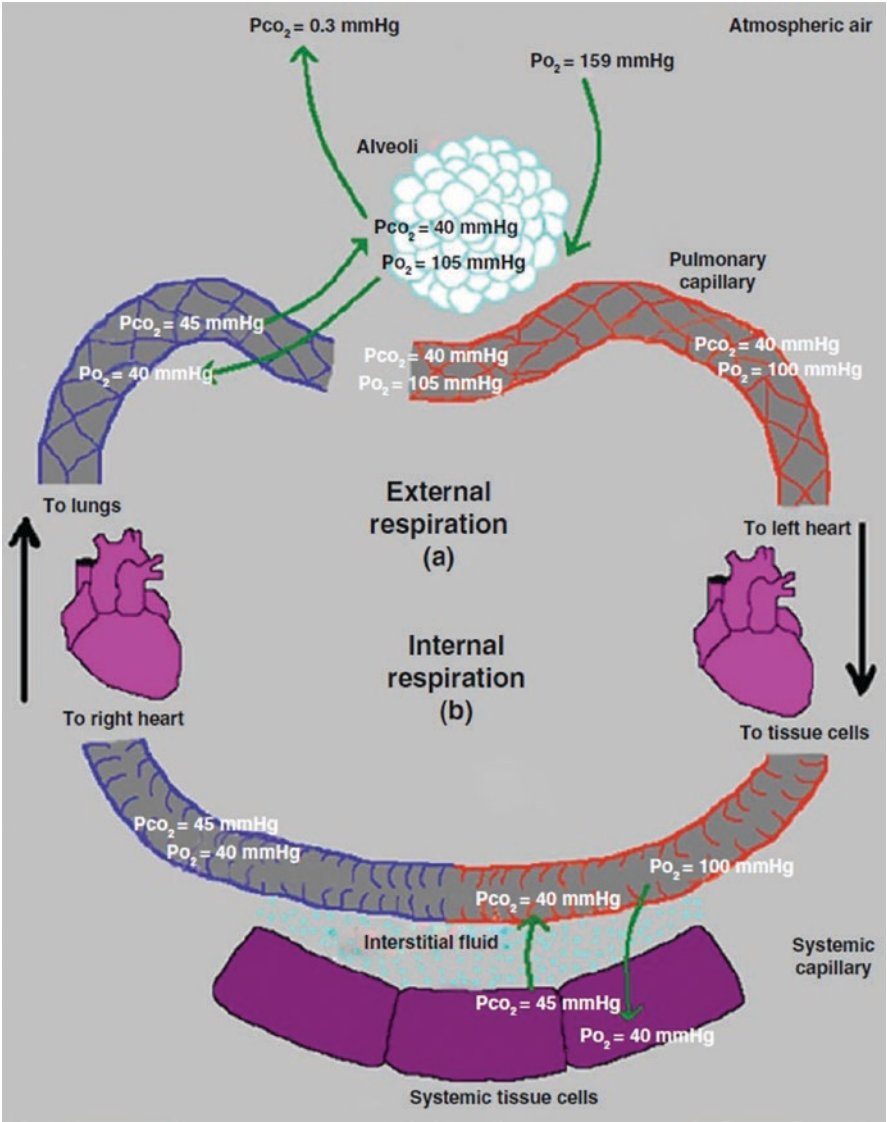
Because of the different partial pressures between the alveoli and the venous blood entering the lungs there is a flow of oxygen from the lung sacs into the blood and conversely a flow of carbon dioxide in the opposite direction. A similar exchange occurs at the tissue level where the  $PO_2$  is less than 40 mmHg and the  $PCO_2$  is 45 mmHg. Gases flow to the cells from oxygenated blood as shown in Fig. 3.4.

Table 3.1 Partial pressures of gases in the alveoli

	Percentage (%)	Partial pressure (mmHg)	Partial pressure (kPa)
Nitrogen	79	563.27	75.10
Oxygen	16	114.10	15.21
Carbon dioxide	5	35.66	4.75

Table 3.2 Blood gas partial pressures

	Venous blood pressure (Pv) in		Arterial blood (Pa) pressure in	
	mmHg	kPa	mmHg	kPa
O <sub>2</sub>	40	5.33	100	13.33
CO <sub>2</sub>	45	6.00	40	5.33



**Fig. 3.4** Oxygen levels in the blood and tissues (Reproduced with permission, Pneupac Ventilation, Smiths Medical International, Luton, United Kingdom)

### 3.4.2.2 How Ventilation Affects the Concentration of Gases in the Alveoli: The Alveolar Air Equation

A key feature of the gas exchange in the alveoli is that the composition of the alveolar air changes only very slightly during resting external respiration despite the fact that de-oxygenated blood enters the pulmonary capillaries via the pulmonary artery with a  $PO_2$  of about 40 mm. While in contact with the alveoli this rises to between 90 and 100 mm. The reason for the stability of the partial pressures of the gases in the alveoli can be explained by the fact that the alveoli are being ventilated carbon dioxide is being removed and oxygen provided. At any one moment, oxygen and carbon dioxide are present together in the alveoli. The actual concentration of oxygen available to the blood is governed by the fraction of oxygen in the inspired air the partial pressure of oxygen and carbon dioxide and the ventilation of the alveoli. This relationship is governed by the alveolar air equation shown in a simple form in Box 3.3. This equation has very important consequences for artificial ventilation and for type 2 respiratory failure (see Chap. 4) where the passage of gas in and out of the alveoli fails and hypoxia results.

#### Box 3.3 The Alveolar Air Equation

The concentration of oxygen in the alveoli in contact with the blood is given by the alveolar air equation. There are a number of versions of this but the simplest to understand is as follows:

$$PaO_2 = PiO_2 - PaCO_2 / R$$

where  $PaO_2$  and  $PaCO_2$  are the partial pressures of oxygen and carbon dioxide in the alveoli, and  $PiO_2$  is the partial pressure of oxygen in the inspired gas.  $R$  is the respiratory quotient which has normal value of 0.8.

The level of  $CO_2$  is itself proportional to the amount of gas passing in and out of the alveoli (alveolar ventilation) according to the following equation:

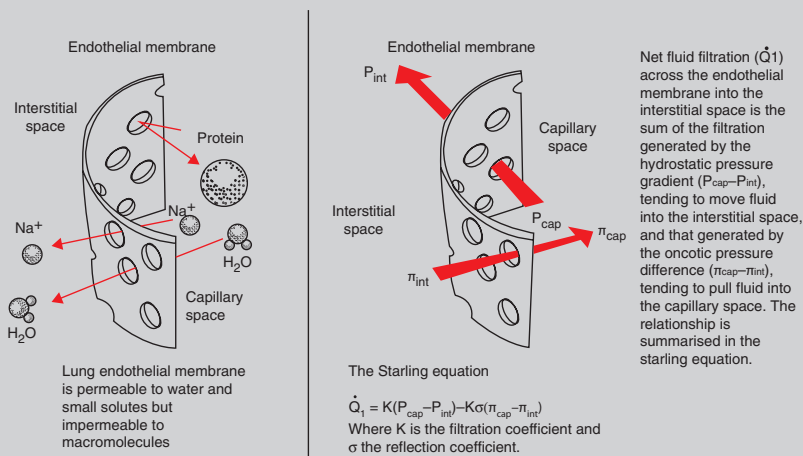
$$PeCO_2 = \dot{V}CO_2 \times k$$

where  $\dot{V}CO_2$  is the amount of carbon dioxide released into the alveoli per minute and  $k$  is a constant. If the  $CO_2$  could be removed from the expired gas by absorption onto soda lime (as in the case with certain types of anaesthesia) hypoxia does not occur provided the minimum oxygen requirement is provided. However this is not possible in emergency ventilation and so an adequate level of ventilation must be maintained to remove the  $CO_2$  produced.

Oxygen and carbon dioxide pass across the alveolar membrane by diffusion and the thickness of the membrane usually presents no barrier. However the partial pressure gradient between the alveoli and capillaries can be affected by changes in ventilation as well as variations in the inspiratory oxygen concentration. As ventilation decreases this gradient becomes lower, while as it increases it becomes greater but only up to a certain point. Increasing inspiratory oxygen concentration is a more effective way of increasing the partial pressure gradient and this is the basis for the treatment of many pathological conditions of the lung through oxygen therapy (Box 3.4).

For carbon dioxide, the gradient is lower than that of oxygen and is about 4–5 mm. Since  $\text{CO}_2$  diffuses about 20 times faster than oxygen at the same partial pressure difference and the speed of diffusion is twice that of oxygen. Thus the exchange of  $\text{CO}_2$  is less affected by alveoli membrane factors than that of oxygen.

### Box 3.4 The Formation of Fluid in the Alveolar Interstitial Spaces



The practical importance of the Starling Equation is that it explains the formation of excess fluid in the alveoli and interstitial spaces, a condition known as pulmonary oedema which is an important indication for artificial ventilation in some cases (see Sect. 4.4.1). This causes a failure of oxygenation of the blood by either cardiac failure or toxic inhalation. The balance of fluid in the interstitial spaces is controlled by a balance of blood pressure in the capillaries and the osmotic pressure in the tissue fluid. These are linked in the Starling equation which shows why, if there is a change in the capillary vs interstitial pressure or a change in the osmotic properties of the capillary endothelium (as a result of exposure to inhaled toxic substances) the fluid levels in alveoli and interstitial spaces will increase.



### 3.4.2.3 The Ventilation Perfusion Ratio

Effective gas exchange in the lungs depends on the close matching of the ventilation of the alveoli with the flow of blood through the pulmonary capillaries. This is expressed as the ventilation—perfusion ratio ( $V/Q$ ). Ventilation perfusion mismatch leads to blood not being sufficiently oxygenated. This may be due to conditions that fill the alveoli with fluid (pulmonary oedema) or a change in the mechanics of the lungs. There are many causes of  $V/Q$  mismatch but from the standpoint of artificial ventilation it is the failure of the flow of gas in and out of the alveoli (alveolar ventilation) or the failure of diffusion of oxygen across the alveolar—capillary membrane which are important and lead to the two types of respiratory failure described in Chap. 4. Figure 3.5 gives an overview of the relationship between ventilation and perfusion.

## 3.5 Oxygen and Carbon Dioxide Transport in the Blood

Oxygen and carbon dioxide are both carried in the blood but the way in which they are carried is very different. Both gases dissolve in the blood and the amount dissolved is proportional to the partial pressure of gases in the air with which the blood is in contact in the alveoli. The amount of any gas dissolved in a liquid in contact with a mixture of gases is proportional to the partial pressure of that gas, as stated by Henry's Law. However, the solubilities of oxygen and carbon dioxide are very different. Carbon dioxide is about 24 times more soluble than oxygen in the blood at any given temperature and partial pressure. This means that only 0.3 ml of oxygen and 3 ml of carbon dioxide are dissolved in the blood. These amounts are far too small to ensure that the supply of oxygen to the cells of the body and the removal of carbon dioxide is maintained. To ensure this, the blood has special carrying mechanisms for both oxygen and carbon dioxide which are supplementary dissolved gases.

Oxygen binds reversibly with the haemoglobin in the blood which greatly increases the oxygen carrying capability. Haemoglobin is made up of a protein, the globin and a pigment containing ferrous iron called haem with which the oxygen reversibly binds. The binding of oxygen to haemoglobin is essential to the understanding of how oxygen is delivered to the tissues of the body.

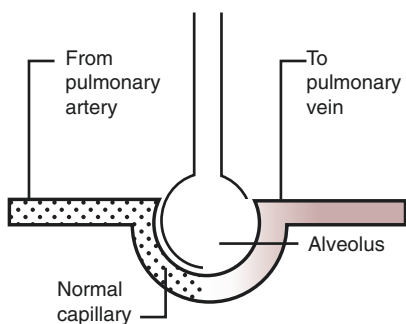
### 3.5.1 The Haemoglobin Dissociation Curve

The binding mechanism for oxygen in haemoglobin is very efficient, there being four binding sites for each molecule. The binding capacity of haemoglobin is expressed in terms of saturation. 1 g of haemoglobin can maximally bind to 1.36 ml of oxygen. At this point the haemoglobin is defined to have a saturation of 100%.

Effective gas exchange depends on the relationship between ventilation and perfusion, expressed as the  $V/Q$  ratio. The illustrations below show what happens when the  $V/Q$  ratio is normal and abnormal.

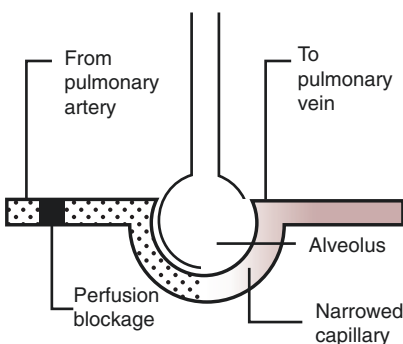
### NORMAL VENTILATION AND PERFUSION

When the  $\dot{V}/\dot{Q}$  ratio is matched, unoxygenated blood from the venous system returns to the right ventricle through the pulmonary artery to the lungs, carrying carbon dioxide. The arteries branch into the alveolar capillaries, where gas exchange occurs.



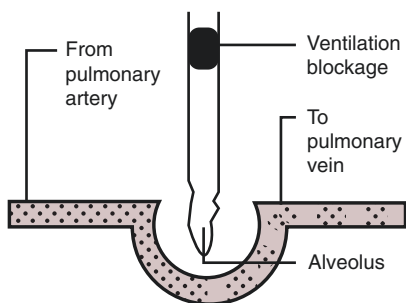
### INADEQUATE PERFUSION (DEAD-SPACE VENTILATION)

When the  $\dot{V}/\dot{Q}$  ratio is high, ventilation is normal but alveolar perfusion is reduced or absent (illustrated by the perfusion blockage). This results from a perfusion defect, such as pulmonary embolism or a disorder that decreases cardiac output.



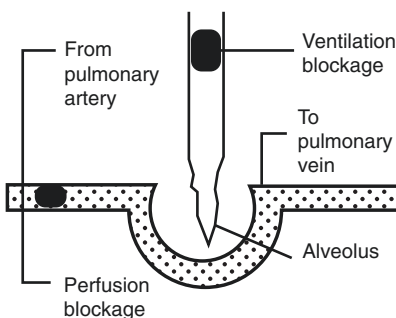
### INADEQUATE VENTILATION (SHUNT)

When the  $\dot{V}/\dot{Q}$  ratio is low, pulmonary circulation is adequate, but oxygen is inadequate for normal diffusion (illustrated by the ventilation blockage). A portion of the blood flowing through the pulmonary vessels doesn't become oxygenated.



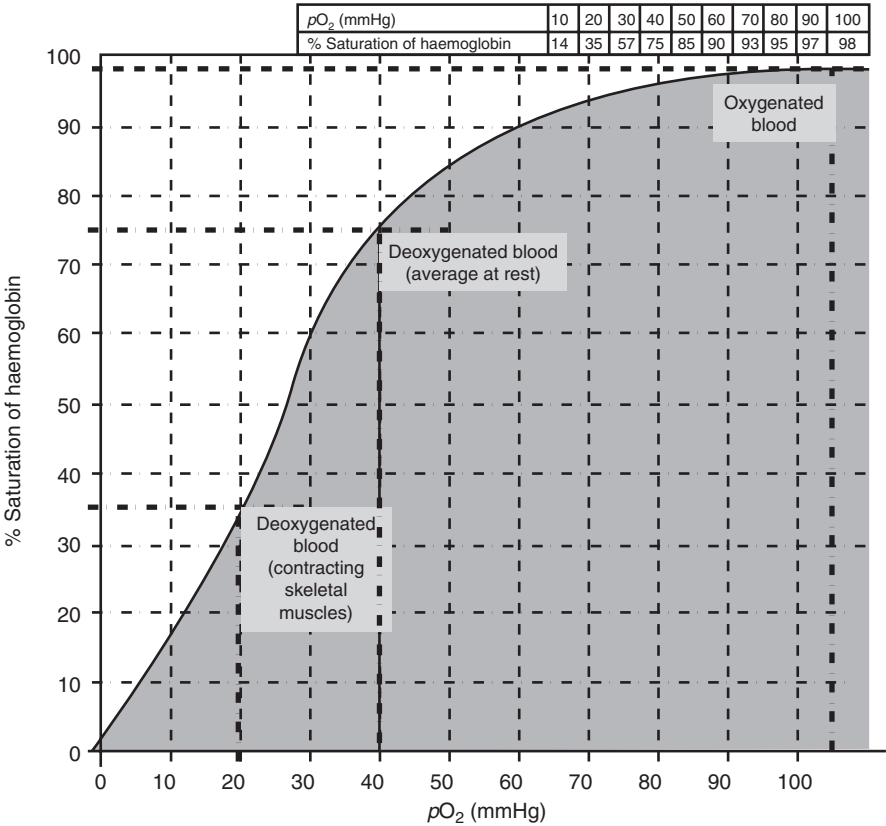
### INADEQUATE VENTILATION AND PERFUSION (SILENT UNIT)

The silent unit indicates an absence of ventilation and perfusion to the lung area (illustrated by blockages in perfusion and ventilation). The silent unit may try to compensate for this  $\dot{V}/\dot{Q}$  imbalance by delivering blood flow to better-ventilated lung areas.



**Fig. 3.5** Ventilation and perfusion of the lungs (Reproduced with permission from Merkle CJ (ed). *Handbook of pathophysiology*. 2nd ed. Philadelphia: Lippincott, Williams and Wilkins; 2005)

The amount of oxygen carried in blood that is 100% saturated is dependent on the haemoglobin concentration in the blood and is calculated by multiplying this concentration in g/dl by 1.36 ml/g. Both oxygen concentration and oxygen content rise in relation to the partial pressure of the oxygen in the air being breathed. Normally this is 21% but the way that oxygen binds to haemoglobin means that the blood is almost fully saturated even at this low proportion of inspired oxygen. The relationship between the partial pressure of the oxygen in the inspired air and the oxygen saturation of the blood is given by the characteristic sigmoid shape of the haemoglobin dissociation curve shown in Fig. 3.6. From this curve the values of oxygen content can be calculated using the haemoglobin concentration and the amount of oxygen in physical solution. Note that the oxygen content is valid only for a given haemoglobin concentration, which determines the height of the curve. Figure 3.6 also shows that as oxygen is removed from the blood the saturation falls so that in venous blood the saturation is characteristically about 75%. Greater degrees of desaturation are found where oxygen usage is higher than usual, as in the



**Fig. 3.6** The haemoglobin dissociation curve (Reproduced with permission, Pneupac Ventilation, Smiths Medical International, Luton, United Kingdom)

case of exercising skeletal muscle. The position of the dissociation curve can shift along the x axis depending on the pH of the blood. In acidosis the curve is shifted to the right which improves the pO<sub>2</sub> for any given saturation level. Conversely, alkalosis (which may be caused by over-ventilation with reduction of  $\text{P}_{\text{eCO}_2}$ ) causes a shift to the left and lowers the pO<sub>2</sub> level for any given saturation.

## 3.6 Internal (Cellular) Respiration

When oxygenated arterial blood reaches the tissue capillaries oxygen diffuses into the cells of the body, driven by a gradient in tissue oxygen tension. The amount of oxygen available to the cells depends on the concentration of haemoglobin in the blood and thus the arterial oxygen content, cardiac output and regional blood flow. Carbon dioxide, which is produced during cellular metabolism diffuses to the regional capillaries and is then transported back to the lungs via the venous circulation.

When oxygen arrives via the arteries and capillaries at the cells it is used in the mitochondria, the cellular power units, to produce the cellular fuel ATP (Adenosine Triphosphate). CO<sub>2</sub> is a waste product of this biochemical process. At rest the body uses 250 ml of oxygen per minute and produces 200 ml of CO<sub>2</sub>. The ratio of the carbon dioxide produced to oxygen consumed is known as the respiratory quotient (R). As we have seen this constant is important in the alveolar air equation. If normal ventilation of the lungs or transfer of gases across the alveolar capillary membrane fails insufficient oxygen is provided to the mitochondria, a condition known as tissue hypoxia. In this situation the cellular respiration switches to anaerobic metabolism which can still produce ATP but much less efficiently than in the case of aerobic metabolism and with the build up of lactic acid as a by product. This has serious consequences. The avoidance of tissue hypoxia is therefore a fundamental aim of artificial ventilation when normal respiration fails.

## 3.7 Conclusions

This chapter has described the function of the lungs both in terms of how oxygen and carbon dioxide are carried to and from the alveoli and these gases are transported in the blood to the cells of the body. The key learning points are:

1. Definitions of breathing and respiration
2. The normal ventilation of the lungs and the definitions of compliance and airway resistance
3. Understanding lung volumes and capacities and the importance of tidal volume and functional residual capacity for artificial ventilation
4. The regulation of breathing by neural centres of the medulla in the brain

5. External respiration and gas exchange with the blood via the alveolar membrane
6. Composition of the gas in the alveoli and the role of the alveolar air equation
7. Transport of oxygen and carbon dioxide in the blood
8. Internal (cellular) respiration

The failure of normal breathing leads to respiratory failure and tissue hypoxia. The recognition and management of this potentially life-threatening condition are the subject of the following two chapters.

## **Suggestions for Further Reading**

Lumb AB. Nunn's applied respiratory physiology. 8th ed. London: Elsevier; 2016.

West JB. Respiratory physiology: the essentials. 10th ed. Baltimore: Lippincott, Williams and Wilkins; 2017.

# Chapter 4

## Respiratory Failure



### 4.1 Introduction

The previous two chapters have described the structure of the airways and lungs and the mechanisms of normal breathing and gas exchange in the blood. In this chapter we consider how this activity can fail, a condition known as respiratory failure. This will cover the pathophysiology (how normal function is changed by factors that can alter the lung structure and function) and also the recognition of respiratory failure both clinically and by the assessment of lung function and blood oxygenation. Understanding respiratory failure is an important stage in deciding whether artificial ventilation is required and also how best to provide it.

Respiratory failure (RF) occurs when the lungs cannot maintain adequate arterial oxygenation or eliminate  $\text{CO}_2$  from the body. This leads to inadequate levels of oxygen in the body cells (tissue hypoxia) where the mitochondria cannot function correctly. Respiratory failure may be acute (ARF) and constitute a medical emergency or be chronic (CRF) as a result of progressive lung disease such as chronic bronchitis. Chronic RF is often exacerbated by superimposed acute episodes. These are termed 'acute on chronic respiratory failure.' Clinically, both acute and chronic RF have characteristic signs and symptoms together with specific blood gas disorders where there are changes in the arterial blood values from those described in the previous chapter. RF usually means a  $\text{pO}_2$  of less than 60 mm Hg and a  $\text{PaCO}_2$  greater than 50 mm. The normal values are 104 and 40 mm respectively in arterial blood (Box 4.1). However, in reality the definition of RF in terms of blood gas values is relative and depends on the patient's medical history. Patients with long-term respiratory disorders such as chronic obstructive airway diseases run with a high  $\text{PaCO}_2$  and a low  $\text{paO}_2$ . In this situation superimposed ARF is indicated by a sudden deterioration in the arterial blood gas values.

**Box 4.1 Arterial Blood Gases**

Although RF and ARF can be recognised clinically, arterial blood gas analysis is very helpful in establishing the diagnosis and the type of failure. Previously only available in hospital laboratories, intensive care units and operating theatres it is a technique that is now used in emergency situations including pre-hospital care. Arterial blood gas analysis (ABG) provides definitive measurements of  $\text{PO}_2$ ,  $\text{PaCO}_2$ , oxygen saturation, the acidity of the blood (pH) and bicarbonate levels.

Normal values for arterial blood gases while breathing air are:

pH 7.35–7.45

$\text{pCO}_2$  4.3–6.0 kPa (32–45 mm Hg)

$\text{pO}_2$  10.5–14 kPa (79–105 mm Hg)

\*Base excess  $\pm$  2 mmol/L

$\text{HCO}_3$  22–26 mmol/L

$\text{O}_2$  saturation 95–100%.

(\*Base excess, which is positive in alkalosis and negative in acidosis is the amount of acid or base which would restore 1 litre of blood to normal acid–base composition at a  $\text{pCO}_2$  of 40 mm Hg).

## 4.2 Definition of Respiratory Failure

There are a number of ways of classifying RF, often based upon the underlying disease. A simple way of understanding RF is to divide it into two forms known as Type 1 and Type 2.

### 4.2.1 Type 1 Respiratory Failure

Type 1 RF is caused (1) by a failure of oxygen to diffuse from the alveolar gas to the pulmonary capillaries (2) by a ventilation/perfusion mismatch (see Chap. 3) or right to left cardiac shunts (e.g. with congenital abnormalities). Typical conditions causing Type 1 RF are pulmonary oedema (both from toxic inhalation and cardiac failure), pneumonia and asthma. It is also a feature of acute respiratory distress syndrome (ARDS) which has many causes, both traumatic and toxic (see Chap. 11).

In type 1 RF the  $\text{PaO}_2$  is low ( $<50$  mm Hg) and the  $\text{PCO}_2$  is normal or low. In this situation the concentration of hydrogen ions in the blood may fall (i.e. an increase in pH), a condition known as respiratory alkalosis.

### **4.2.2 Type 2 Respiratory Failure**

Type 2 RF is caused essentially by hypoventilation either by a failure of the central nervous drive or mechanical failure of breathing or from obstruction to the flow of gas through the airways (asphyxiation). This form of respiratory failure is caused by a failure of ventilation of the alveoli causing an increase in the alveolar  $p\text{CO}_2$ . This causes the  $p\text{O}_2$  in the alveoli to drop as described by the simple alveolar air equation (see Chap. 3). Because the  $p\text{CO}_2$  rises and the gas diffuses back into the blood the concentration of hydrogen ions in the blood (given by the pH) also rises with a drop in the pH. This condition is known as respiratory acidosis. In condition the kidney compensates for the dangerous acidity in the blood by retaining bicarbonate which causes pH to rise (the concentration of hydrogen ions to fall). In type 2 RF the  $\text{PaO}_2$  is low ( $<50$  mm Hg) and the  $\text{PaCO}_2$  is high ( $>50$  mm Hg). Type 2 respiratory failure may be partial or complete (respiratory arrest).

### **4.2.3 Acute Respiratory Failure**

In some situations, notably respiratory epidemics the non-specialist in artificial ventilation is likely to have to deal with acute respiratory failure (ARF) or acute on chronic respiratory failure and it is important to be able to recognise these quickly. Overall, ventilation–perfusion inequalities are the most important cause of low  $\text{PaO}_2$  in ARF, complicating previous disease conditions whereas hypoventilation and acute pulmonary oedema (filling of the alveoli with fluid, caused by cardiac dysfunction or inhalation of toxic gases such as chlorine) are the main causes of ARF in previously normal lungs. Note that this view of ARF includes both type 1 and type 2 respiratory failure.

Note also that both hypoventilation and V/Q inequality can give rise to  $\text{CO}_2$  retention but hypoventilation is the essential cause of type 2 respiratory failure whereas V/Q inequality is the cause in long-standing chronic obstructive airways disease and interstitial disease such as ARDS.

## **4.3 Tissue Hypoxia**

During both types of RF there is a reduction in the normal level of oxygen in the cells of the body, a condition known as tissue hypoxia. Reduction of oxygen levels in the cells leads in turn to a reduction at the important site of the mitochondria (Box 4.2) where oxygen is used to provide energy during aerobic metabolism. The reversal of tissue hypoxia is the key aim of artificial ventilation.



**Box 4.2 Effects of Hypoxia on the Mitochondria of the Cells Mitochondrial Function**

When the tissue  $\text{PO}_2$  falls below a critical level, normal aerobic metabolism producing ATP which fuels the body cells ceases and is replaced by anaerobic metabolism which still produces ATP but much less efficiently. The critical value of tissue  $\text{pO}_2$  at which this happens is not known but is thought to be around 1 mm Hg. If tissue oxygenation improves following artificial ventilation for ARF the lactic acid which builds up during anaerobic metabolism can be reconverted to glucose or used directly in the liver to provide energy.

There are several factors which contribute to tissue hypoxia. These include low arterial oxygen tension ( $\text{PaO}_2$ ), the oxygen capacity of the blood, Hb affinity for oxygen, cardiac output and distribution of blood flow (e.g. in hypovolaemic shock following blood loss, blood is transferred to the central organs away from the skin).

**4.3.1 Vulnerability of Various Body Tissues to Hypoxia**

The cells of the body have different vulnerabilities to hypoxia. The cells at greatest risk are:

1. Neurones in the brain: Cessation of the blood flow to the cerebrum causes loss of function within 4–6 s, loss of consciousness within 10–20 s and irreversible changes leading to brain death within 3–5 min.
2. Myocardium: The muscle cells of the heart are also very vulnerable to tissue hypoxia which ultimately leads to cardiac arrest. Note that this type of cardiac arrest is secondary to ARF, unlike primary cardiac arrest which is caused by occlusion of the coronary circulation and interruption of blood flow. It is the main cause of cardiac arrest in infants and children.

The vulnerability of these two systems to hypoxia is the basis of early intervention and artificial ventilation following primary respiratory arrest.

**4.4 Causes of Respiratory Failure**

A large number of conditions can lead to respiratory failure and various classifications are possible. Box 4.3 shows an overall classification for both acute and chronic respiratory failure.

**Box 4.3 Causes of Respiratory Failure****Traumatic**

- disruption of breathing mechanisms through physical trauma
- disruption of cerebral control mechanisms-raised intracranial pressure, brainstem injury

**Infectious**

- acute (viral and bacterial) and chronic (e.g. COAD) infections
  - effects on neural control
  - effects on lung parenchyma
  - V/Q mismatch (from ARDS)

**Circulatory**

- cardiac pulmonary oedema (heart failure)

**Neurological**

- acute exacerbation of myasthenia gravis

**Toxic**

- Toxic trauma to the airways and alveoli
- Toxic pulmonary oedema
  - Paralysis of respiratory muscles and suppression of the respiratory centres of the medulla of the brain (e.g. from organophosphate poisoning).

**4.4.1 Pathophysiology of Type 1 and Type 2 Respiratory Failure**

The main causes of type 1 acute respiratory failure are:

- Cardiogenic pulmonary oedema (due to heart failure)
- Pneumonia
- Chronic obstructive airway disease (COAD) exacerbation
- Pulmonary embolism
- ARDS from numerous causes (see Chap. 11)
- Toxic pulmonary oedema (following the inhalation of very high concentrations of toxic gases)

The main causes of type 2 respiratory failure are:

- Respiratory centre depression (traumatic and toxic), leading to a depression or failure of breathing
- COAD

- Asthmatic crisis
- Muscle weakness (e.g. acute Guillain Barre syndrome, myasthenia gravis)
- Chest trauma and deformities
- Paralysis of respiratory muscles by chemical agents and toxins

The most common cause of ARF is airway obstruction which can be acute (e.g. foreign body blocking the airway) or following an acute exacerbation of COAD.

## 4.5 Consequences of Acute Respiratory Failure

Hypoxaemia and hypercapnia which are characteristic of ARF trigger responses in all the body systems, including the respiratory, central vascular system, central nervous system and the kidneys. For example, in the autonomic nervous system the sympathetic section triggers vasoconstriction which causes increased peripheral resistance and increased heart rate.

Tissue hypoxia occurs as a result of ARF causing anaerobic metabolism and a rise in lactic acid levels leading to acidosis. This respiratory acidosis is caused by a build-up of  $\text{CO}_2$  and is signalled by an increase in heart rate and stroke volume.

Cyanosis (a blue coloration of the skin and mucous membranes) is apparent when there are increased concentrations of de-oxygenated haemoglobin in the blood. Note that the concentration of Hb must be above 5 gm/100 ml for cyanosis to be recognised. If respiratory failure is accompanied by major loss of blood in trauma this may not be the case.

Hypoxia of the kidneys causes a release of the hormone erythropoietin from the renal cells which stimulates the bone marrow to increase red cell production to attempt to improve the oxygen carrying capability.

Overall in ARF there is a somatic response to the build up of  $\text{CO}_2$  with cerebral depression, hypotension, circulatory failure, followed by increased heart rate and cardiac output.

The effects of hypoxia and hypercapnia on the brain are important since the neurones of the brain are the most sensitive cells in the body to hypoxia. Both hypoxia and hypercapnia act on the respiratory centre in the medulla to (1) increase breathing depth and therefore the tidal volume (2) increase respiratory rate. This is followed by recruiting muscles which are not normally involved in breathing (the accessory breathing muscles) to assist normal respiratory muscles to try and improve the depth of breathing.

## 4.6 Acute on Chronic Respiratory Failure

Patients who are in chronic respiratory failure are chronically hypoxic and have high levels of  $\text{CO}_2$ . A classic example of this situation is chronic obstructive airways disease (COAD). In this situation the main stimulus to breathing in the respiratory centre comes not from raised  $\text{CO}_2$  which is the case in normal breathing but from

blood hypoxia which is detected by other systems located in the main arteries. The signals from these receptors keep breathing going by hypoxic drive.

The importance of remembering hypoxic drive in the management of acute ARF in a patient with chronic ARF is that if the  $\text{PaO}_2$  is suddenly increased as a result of breathing high concentrations of oxygen through a mask the hypoxic drive will be lost and the patient will stop breathing. For this reason delivered oxygen concentrations to patients with COAD are kept at a maximum of 40%. In extreme cases if this concentration is not sufficient to correct tissue hypoxia the patient will require artificial ventilation with a higher concentration of oxygen. For other reasons however long term ventilation with oxygen is not wise due to the potential toxicity of oxygen itself causing type 1 respiratory failure.

## **4.7 Recognising Respiratory Failure**

### ***4.7.1 Introduction***

Artificial ventilation in emergency is required when the patient's own breathing efforts are inadequate, leading to partial or total respiratory failure. Assessment of breathing is an essential part of the rapid primary assessment and subsequent secondary assessment of an emergency. Thus, recognition of breathing failure and the need for airway and ventilation support must be integrated closely with the other sections of the guidelines for basic and advanced life support. These are published internationally and reviewed every 5 years by the International Liaison Committee on Resuscitation (ILCOR) an organisation which links resuscitation councils around the world, such as the European Resuscitation Council and the American Heart Association. Such guidelines are evidence-based and are taught by practical skill sessions at a basic and advanced level. The guidelines will provide the basis for the practical management of artificial ventilation covered in subsequent chapters.

### ***4.7.2 Assessment of Breathing: General Considerations***

In an emergency patient survey (airway, breathing, circulation, disability and exposure - ABCDE) airway, breathing and level of consciousness affect the ventilation of the lungs and the development of hypoxia. From the outset, it is important to note that breathing can only take place if the airway is open but an open airway does not necessarily mean that breathing and lung ventilation are taking place. The integration of airway and breathing assessment is therefore fundamental to the provision of successful artificial ventilation.

Although resuscitation and advanced trauma life support guidelines follow through the sequence of ABCDE for primary and secondary patient surveys for respiratory emergencies the AB and D are assessed almost simultaneously.

4.7.3 *Assessment of Conscious Level*

The ILCOR guidelines recognise that establishing the level of consciousness is a vital first stage in CPR by trying to arouse an unconscious patient (‘shake and shout’). Equally, a rapid assessment of conscious level is an important first stage in management of respiratory emergencies since the airway may be rapidly lost during unconsciousness. There are a number of ways of expressing degrees of unconsciousness but the quickest and simplest is the AVPU system. This assesses whether the patient is fully conscious and alert (A), is apparently unconscious but responds to voice commands (V), responds to painful stimuli (P) or is totally unresponsive (U). Another system of assessment is the Glasgow Coma scale which is described in Box 4.4. This system takes longer to assess than AVPU, an important consideration where there is total life-threatening hypoxia as a result of respiratory arrest.

**Box 4.4 The Glasgow Coma Scale**

This scale assesses unconsciousness according to the following observed responses:

Eyes Open	Spontaneously	4
	To verbal command	3
	To painful stimulus	2
No response		1
Best verbal response		
	Orientated and converses normally	5
	Disorientated but converses	4
	Inappropriate words	3
	Incomprehensible sounds	2
	No response	1
Best motor response		
To verbal command	Obeys	6
To painful stimulus		
	Localises pain	5
	Flexion(withdrawal)	4
	Flexion (abnormal)	
	(decorticate rigidity)	3
	Extension	
	(decerebrate rigidity)	2
	None	1
Total		3–15

If the GCS score is equal to or less than eight the patient should have the airway secured and be artificially ventilated.

#### **4.7.4 Airway**

The unconscious patient who does not respond to vocal or painful stimuli is at great risk from airway obstruction. The anatomy of the upper airway, described in Chap. 2 showed that the greatest risk of airway obstruction comes from the tongue. Man is one of a limited number of species whose airway becomes blocked during unconsciousness (i.e. with the loss of airway protective reflexes) as opposed to normal sleep, where the reflexes are preserved. In addition the airway may be blocked in a conscious person by secretions, vomitus and blood in the case of trauma. Securing the airway is done by positioning the patient and the head, and the use of primary and secondary airway devices. These are described in the next chapter.

#### **4.7.5 Primary Breathing Assessment**

Normal breathing in a conscious person at rest is largely provided by the diaphragm which moves up and down under reflex control from the medulla of the brain. The intercostals muscles which control the shape of the thorax are usually employed during exercise when breathing increases.

Breathing is assessed by:

*Looking* at the patient (inspection)

*Listening* (at both the mouth and the chest, using a stethoscope)

In normal breathing, inspection shows an alert patient, the colour of the lips and tongue is normal and there is no evidence of cyanosis except where there is a history of pre-existing lung disease (e.g. chronic bronchitis). Movements of the chest are normal and there is no see-saw movement in relation to the abdominal muscles. The accessory respiratory muscles (see Chap. 2) are not used.

Listening to air intake at the mouth reveals no major sounds of obstruction (equivalent to snoring during normal sleep). There is no wheezing (caused by contraction of the smooth muscle around the bronchioles).

In *abnormal breathing* there may be (1) airway blockage (2) failure of breathing or both. When the airway is blocked but breathing efforts are still present there is paradoxical breathing (where the chest and abdomen move in a see-saw motion). There is use of the accessory muscles of respiration which are not normally used and evidence of tracheal tug. In a rapid primary assessment, the stethoscope is used to assess whether air is entering the right and left main bronchi equally or not at all.

#### **4.7.6 Secondary Patient Assessment**

The assessment of the respiratory status in the secondary survey involves looking and listening, as above with more extensive auscultation and also taking the history of the presenting problem if possible. This is an essential first step if the patient is conscious and there is no immediate life-threatening situation.

Questioning should include

1. What are the circumstances of the emergency? (is there evidence of physical trauma or the release and inhalation of a toxic substance (toxic trauma))
2. How quickly has the respiratory difficulty developed?
3. Any history of pre-existing chest problems (chronic bronchitis, asthma)?
4. Any history of respiratory infection? (cough, chest pain, production of sputum (colour, viscid or frothy))
5. Smoking history and history of any possible exposure to inhaled toxic substances (did the patient see or smell anything suspicious?)

#### 4.7.6.1 Hyper – and Hypo- Ventilation

If the airway is open and the patient is conscious assess the adequacy of ventilation. This is determined by the breathing rate and the tidal volume  $F \times V_t = V_m$  (the minute volume). The minute volume should normally be 3–6 litres/min in an adult at rest. As noted earlier, each breath consists of dead space and air that takes an active part in gas exchange in the alveoli. In an initial assessment it is usually possible only to assess the rate and depth of breathing visually. A rate of breathing <8 or >20/min indicates respiratory failure.

Hyperventilation describes a patient who is breathing too fast. However, if the breaths are too shallow there will not be adequate alveolar ventilation and the patient will in effect be hypo-ventilating since the minute volume will not be adequate to ensure correct oxygenation of the blood. This type of breathing is common in trauma and leads to a build up of  $CO_2$  (type 2 respiratory failure—see Chap. 3). If the patient is hyperventilating with an adequate tidal volume the  $CO_2$  level will be reduced, as in the condition of hysterical hyperventilation.

Hypoventilation occurs if the breathing rate is too low although the tidal volume may be adequate. This situation occurs in emergency from head injury trauma and also from the respiratory centre depression from opiate overdosage. Note that if the patient is in pain normal doses of opioids required to control the pain will not usually cause hypoventilation. In hypoventilation the minute volume is reduced. This can occur either as a reduction in rate of breathing, a reduced tidal volume or both.

In addition to hyper- and hypo-ventilation which are observable signs, the patient, if conscious may complain of difficulty in breathing or dyspnoea (breathlessness). This is a common symptom in both chronic respiratory disease and also respiratory emergency such as pneumonia and ARDS. It can equally indicate respiratory conditions such as asthma or chronic obstructive airway disease but also non-respiratory conditions such as cardiac failure and severe anaemia.

## **4.8 Aids to the Clinical Assessment of Respiratory Failure**

### **4.8.1 Auscultation**

So far the discussion of the assessment of respiratory failure has depended on the use of basic clinical skills such looking and listening. For the latter the stethoscope can be used by those suitably trained in a primary assessment role to assess air entry into the lungs. For a secondary assessment the key sounds of auscultation will provide an indication of the cause of respiratory failure. This involves listening to the chest over the main airway levels and also at the bases of the lungs posteriorly.

The key sounds to listen for are:

1. Rhonchi: a low pitched sound indicating large airway obstruction from mucus or inhaled secretions
2. Wheezing: a higher pitched sound indication small airway obstruction by spasm of the muscle fibres around the bronchi as in the case of an asthmatic crisis
3. Crackling sounds at the bases of the lungs indicating the presence of fluid in the alveoli from pulmonary oedema or pneumonia

### **4.8.2 Pulse Oximetry**

Although the detection of cyanosis in the primary survey can give a good indication of the degree of oxygenation of haemoglobin (if the level is sufficiently high) the saturation level of haemoglobin can be measured directly by using the technique of pulse oximetry. Originally a technique that could only be used in the operating theatre and ICU, pulse oximetry devices have now been made small, portable and easy to use, making the technique a valuable addition to emergency management outside the hospital.

#### **4.8.2.1 Principle of Operation**

Pulse oximetry depends upon the absorption of light at a specific frequency when passed through a suitable anatomical site containing a capillary bed. A finger or ear lobe are common sites. A pulse oximeter (Fig. 4.1) contains light-emitting diodes, a photocell detector with a microprocessor control and a visual display unit. The principle of the device is based upon the different absorption of light by oxygenated and deoxygenated haemoglobin at a wavelength of 660  $\mu$ .



**Fig. 4.1** The Digit finger pulse oximeter (Photograph courtesy of Smiths Medical International, Luton, United Kingdom)



Essentially therefore, the pulse oximeter measures the variation related to the arterial pulse, in absorption of a light beam passed through the blood. The device can therefore provide information about the pulse rate and also the oxygen saturation of the blood, by calculation of the absorption of light by oxygenated Hb and cancelling out the effects of other tissues, venous blood and background light.

Pulse oximetry is accurate to within 2% but this falls to 5% with saturations below 80%. The factors affecting accuracy are:

1. *Smoking*: This overestimates the saturation by the percentage of carboxyhaemoglobin present (this ranges between 3% in urban dwellers to 15% in heavy smokers)
2. The presence of *methaemoglobin* (often used for the treatment of cyanide poisoning) where the saturation tends towards 85%
3. *Cardiac dyes*: Used to determine cardiac output, which can cause an underestimation
4. *Extraneous light*, movement and diathermy can all affect absorption. Modern pulse oximeters have sophisticated computer control of these artefacts
5. *Atrial fibrillation* and *vasoconstriction* which lead to a poor pulse volume, with errors in measurement.

Pulse oximetry has a number of advantages as a non-invasive continuous monitoring system; it gives a measure of Hb saturation, and early warning of hypoxic events and is easy to use in emergency management. It does not however replace good clinical observation.

### 4.8.3 Measurement of End Tidal $\text{CO}_2$ (Capnography)

In recent years the measurement of the concentration of carbon dioxide in exhaled air has become possible outside the laboratory in the operating theatre and ambulance. The value of capnography which measures  $\text{CO}_2$  concentrations at the end of an expired tidal volume (end tidal  $\text{CO}_2$ ) is that it can give an immediate indication of increased  $\text{CO}_2$  during type 2 respiratory failure and also the effectiveness of artificial ventilation once started. It is also very valuable in checking whether an endotracheal tube has been correctly inserted.

Infra-red light is absorbed by carbon dioxide in a gas flow and this fact is the basis of the determination of carbon dioxide concentration in exhaled air by capnography. Light in the infrared waveband is absorbed by all gases with dissimilar atoms in the molecule. Therefore there are many potential sources of interference with estimating carbon dioxide.

There are two types of capnograph. (1) Mainstream, where a measuring head is placed in the main stream of the gas coming from the ventilated patient and (2) side stream, where a small amount of the exhaled gas is bled off via a sampling line (Fig. 4.2). Mainstream capnographs are more responsive but are difficult to maintain. Thus side stream capnographs are more commonly used. A problem with both devices is the response time. A 90% response is usually obtained within 0.2 s. Most capnographs can cope with respiratory rates of up to 60/s. If however there is a long sampling line inaccuracies can occur at rate of 40/min.

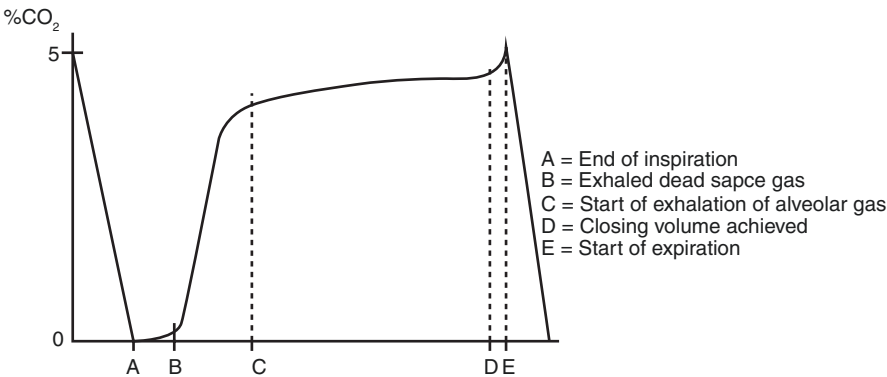
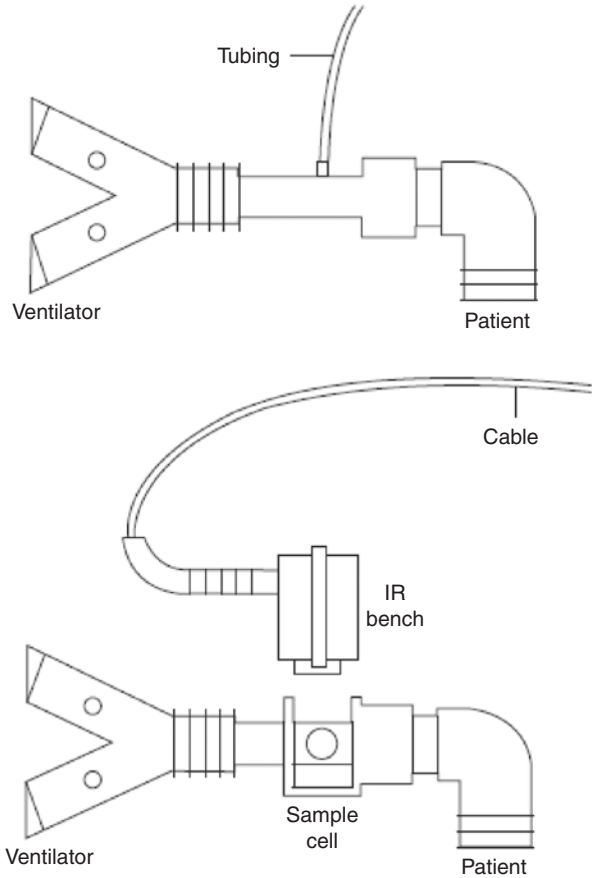
#### 4.8.3.1 Waveform Analysis in Capnography

The analysis of the waveform of the expired  $\text{CO}_2$  in capnography, both from a single spontaneous breath and from continuous monitoring during IPPV can give valuable clinical information. This has been endorsed by the ILCOR in its guidelines for cardiopulmonary resuscitation (see Chap. 5).

##### Single breath waveform

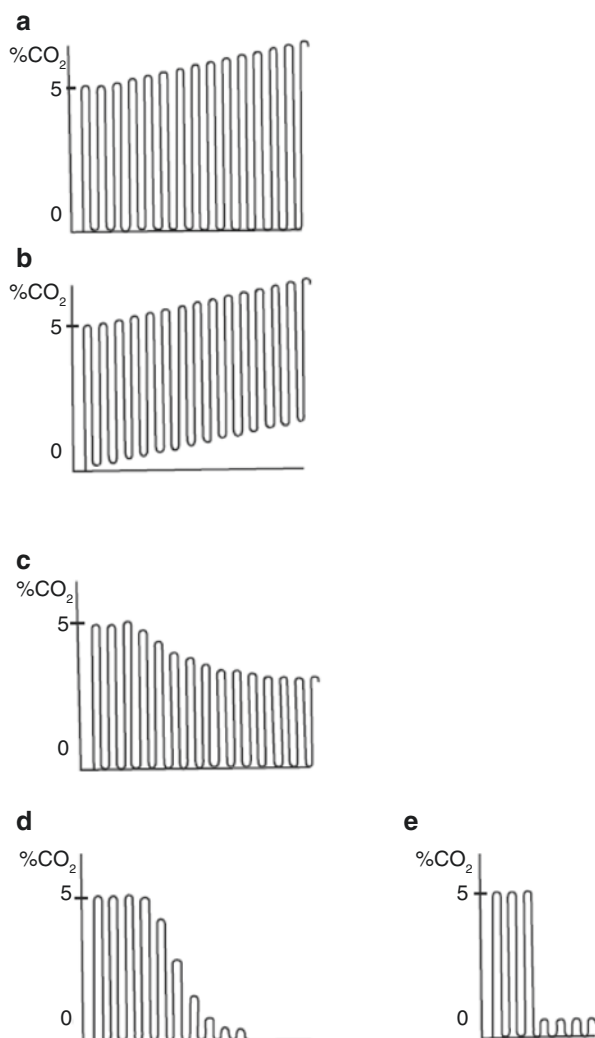
This involves analysis of a single breath with an expanded x axis. This is shown in Fig. 4.3. During expiration the curve between points A and B represents dead space (fresh) gas with almost no  $\text{CO}_2$ . Between points B and C there is a rapid rise in end tidal  $\text{CO}_2$  due to the presence of alveolar gas. The plateau between C and D represents the emptying of the alveoli and is often uneven due to different areas of lung emptying at different rates and because the lung volume is reducing while  $\text{CO}_2$  excretion continues. Point D represents the closing volume where no further gas is exhaled. Point E represents the commencement of the next inspiration. Analysis of the single breath waveform can provide information about underlying pathophysiological conditions such as asthma where there is a steep rise in the curve between D and E and ventilation related conditions such as the patient taking a single spontaneous breath while being ventilated (seen as a dip in the plateau) or rebreathing where the capnographic record does not return to zero on the start of inspiration. This is due to excess dead space in the circuit.

**Fig. 4.2** Side stream (A) and mainstream capnographs



**Fig. 4.3** Single breath capnograph waveform

**Fig. 4.4** Capnography trend analysis. (a) Hypercapnia—carbon dioxide over production or absorption. (b) Hypercapnia—rebreathing. (c) Hypocapnia—overventilation (d) hypocapnia—collapse. (e) 'Hypocapnia'—disconnection



#### Trend analysis

Apart from analysis of a single breath the time base on the x axis can be compressed to show trends in repeated breaths, indicating hypercapnia or hypocapnia. These are shown in Fig. 4.4.

Hypercapnia can be caused by:

- increased CO<sub>2</sub> production eg in fever or reduced CO<sub>2</sub> elimination due to an inadequate minute volume. In the graphs shown in Fig. 4.4 note that the baseline remains on the x axis in this situation.
- re-breathing, during spontaneous breathing in anaesthesia with an inadequate fresh gas flow.

Hypocapnia can be caused by:

- (a) over-ventilation with too high a minute volume
- (b) a major cardiovascular disturbance (e.g. pulmonary embolus or cardiac arrest)
- (c) kinking of the endotracheal tube or disconnection of the circuit

#### **4.8.3.2 Uses of Capnography**

The major uses of capnography are:

1. Confirmation of endotracheal intubation (a correct single breath waveform). Note that in the case of oesophageal intubation a false positive waveform can be observed but this only lasts for the first few breaths
2. Determination of the correct level of minute ventilation during IPPV
3. During anaesthesia, to determine the fresh gas flow
4. To detect circuit disconnection in the case of IPPV (this information supplements the disconnect alarm on the ventilator which is triggered by a drop in inflation pressure)

### **4.9 Conclusions**

1. Respiratory failure (RF) may be defined as the failure of normal breathing and lung ventilation leading to abnormally low levels of oxygen (hypoxia) in the blood and tissues.
2. Respiratory failure may be slow in onset (chronic), rapid (acute) and acute on chronic where there is a sudden degradation of chronic failure.
3. There are a number of ways of classifying RF but in the acute setting a suitable division is (1) type 1, where there is a failure of oxygen to be carried from the alveoli of the lungs to the blood in the pulmonary capillaries and (2) type 2, where there is a failure to ventilate the alveoli leading to a build-up of carbon dioxide.
4. Respiratory failure leads to tissue hypoxia, where the mitochondria in the cells of the body cannot generate energy by aerobic metabolism. Although this can be replaced in the short term by anaerobic metabolism hypoxia is a potentially life-threatening situation.
5. Different organs in the body are more vulnerable to hypoxia than others. The brain and the heart are most at risk.
6. There are a number of causes of RF, both acute and chronic, including traumatic, infectious, circulatory and toxic.
7. Early recognition of RF is important, particularly in emergency. Assessment of the airway, breathing, circulation and conscious level are essential in the rapid

primary examination. In a secondary assessment more information can be gathered from a good patient history and careful clinical examination.

8. Useful aids to the diagnosis of RF include pulse oximetry, capnography and blood gases. These may now be measured in portable form in the prehospital setting.

## **Suggestions for Further Reading**

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## Chapter 5

# The Management of Respiratory Failure: Airway Management and Manual Methods of Artificial Ventilation



### 5.1 Introduction

Non-mechanical methods of ventilation preceded mechanical ventilation and still have an important place in modern medical practice, particularly in resuscitation. Expired air ventilation was probably the first form of artificial ventilation and is still indicated as a primary method of ventilation for cardiac and respiratory arrest. The bag–valve (BV) device, which succeeded the bellows used in the eighteenth and nineteenth century is widely used in both the hospital and prehospital settings around the world and for many emergency medical services remains the only authorised method of AV, particularly in parts of the US. Negative pressure methods of artificial ventilation, developed and used extensively during the 19th and first part of the twentieth centuries were shown to be ineffective and are not now used. This chapter concerns all available methods of non-mechanical artificial ventilation and the essential associated airway protection techniques. Non-mechanical ventilation methods have been assessed using evidence—based techniques by the world regional resuscitation councils that form the International Liaison Committee for Resuscitation (ILCOR). This body produces definitive guidelines for resuscitation every 5 years which cover all aspects of cardiopulmonary resuscitation. The latest guidelines appeared in 2015 and supplemented the previous 2010 guidelines. At the time of writing the guidelines are due to be updated in late 2020. However, with the current COVID19 outbreak there is likely to be a postponement. The following discussion is based on a synopsis of the ILCOR airway management and non-mechanical ventilation guidelines for both basic and advanced life support in adults from 2015. Readers are advised to consult appropriate websites for the latest guidelines (see suggestions for further reading). The equivalent measures for children are discussed in Chap. 9 ventilation using a self reforming bag, although widely—regarded as being safe, has increasingly been recognised as having potential

dangers and these are discussed in detail, based upon published evidence in the second part of the chapter.

Once diagnosis of respiratory failure or arrest has been made immediate steps must be taken to manage it. These comprise (1) management of the airway and (2) provision of oxygen support and artificial ventilation.

### ***5.1.1 The Degree of Respiratory Failure***

There is a spectrum of respiratory failure which extends from mild depression of breathing through to complete respiratory arrest. The type of support required therefore depends on the position of the patient in this spectrum. As will be seen in Chap. 6 (mechanical ventilation) artificial ventilation support can be adapted to the degree of support required and this is usually the case in hospital practice. In emergency however, the degree of ventilation support is usually divided between free flow oxygen therapy, demand ventilation and supportive or total AV.

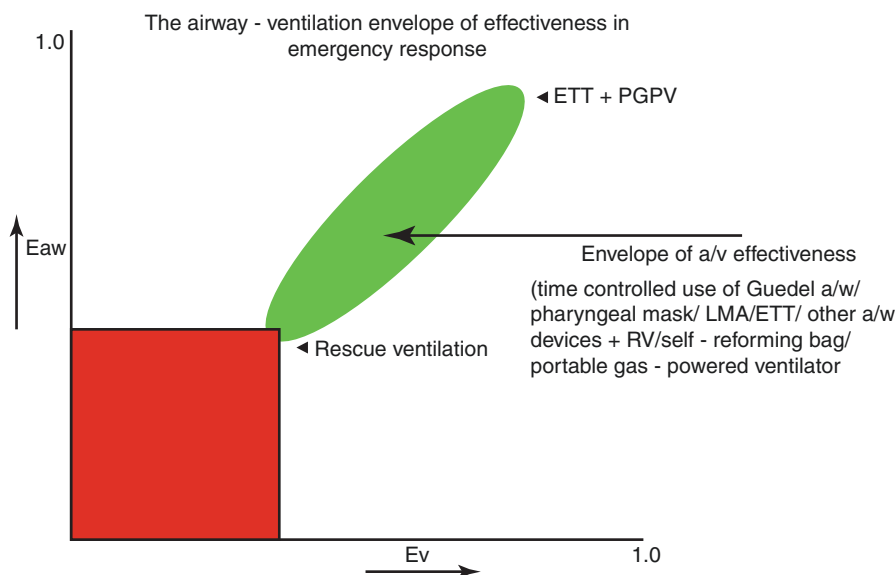
### ***5.1.2 Equipment***

The equipment used in the management of respiratory failure varies in its complexity. It is essential that the equipment used is familiar to operators and adapted to their skill levels and training. There are different skill levels between medical and paramedical personnel and the type of respiratory support provided will depend on who is giving it. Equipment used must be adapted to the circumstances of the emergency response where most of the basic services found in the hospital such as piped oxygen supplies and mains electricity may not be available. In emergency ventilation there must be simplicity of operation and reliability of the method used. However, there is a gradual expansion of the skills and equipment available for emergency ventilation.

### ***5.1.3 Airway: Ventilation Management***

Airway management and the provision of artificial ventilation are usually regarded as separate skills but in practice there must be integration of the two. The endotracheal tube is regarded as being the 'gold standard' of airway management but if too long a time is taken to insert it before ventilation is started the degree of hypoxia produced by respiratory failure may be seriously worsened. In this situation it would have been better to continue using a simple face mask with AV. Increasingly, the laryngeal mask airway is recognised as a more appropriate device for use in emergency by responders with limited experience of endotracheal intubation.





**Fig. 5.1** The airway—ventilation envelope of effectiveness in emergency response. The green area shows effectiveness of difference combinations of airway and ventilation procedures where  $E_v$  = effectiveness of ventilation and  $E_{aw}$  = effectiveness of the airway. The most secure and effective is endotracheal intubation and ventilation with a portable ventilator. However, if there is a delay in establishing intubation this effectiveness is lost and other combinations would have been more effective in overcoming hypoxia. The envelope is a function of availability of equipment, skills in using it and the time taken to commence ventilation

There exists an envelope of airway and ventilation options which can be adapted to the skill levels and circumstances. This is shown diagrammatically in Fig. 5.1.

## 5.2 Management of the Airway During Non-mechanical Artificial Ventilation: General Considerations

A detailed discussion of airway management techniques and how they are taught is outside the scope of this book and the reader is referred to the many specialised texts on the subject which have been written for medical (including emergency physicians, anaesthetists and ICU specialists), paramedical and nursing personnel. The following points are important:

The airway can be opened and kept open by

### 1. Simple positional management

- Head tilt chin lift
- Jaw thrust
- Left lateral position

## 2. Airway adjuncts

- Guedel airway
- Supraglottic airway devices—the LMA in particular
- Endotracheal intubation
- Cricothyroid access—PCK and similar devices

### 5.2.1 *Airway Obstruction*

Unless there is a clear airway ventilation of the lungs cannot take place. Recognition of airway obstruction and its management is therefore fundamental to successful artificial ventilation. Patients requiring resuscitation often have an obstructed airway, usually secondary to loss of consciousness where there is a loss of protective reflexes to keep the airway open. Unless the airway is kept open and ventilation assured airway obstruction will lead to cardiac arrest. This is particularly the case in children where cardiac arrest is usually secondary to primary respiratory arrest. Thus early recognition and assessment, with control of the airway and ventilation of the lungs is essential to prevent secondary hypoxic damage to the brain and other vital organs. Without adequate oxygenation it may be impossible to restore a spontaneous cardiac output. The chances of successful defibrillation of the heart for example, decrease by 10% for every minute of hypoxia in the heart muscle and conducting system.

#### 5.2.1.1 Causes of Airway Obstruction

Obstruction of the airway may be partial or complete. It may occur at any level, from the nose and mouth down to the trachea (Chap. 2). The commonest site of airway obstruction in an unconscious patient is at the soft palate and epiglottis. Unlike most animals, man does not have an automatically protected airway. Obstruction of the nasopharynx and upper airways may also be caused by vomit or blood (regurgitation of gastric contents or trauma), or by foreign bodies. Obstruction at the level of the larynx may be caused by oedema from burns, inflammation or anaphylaxis with oedema of the larynx.

In a patient who is not deeply unconscious stimulation of the nasopharynx may cause laryngeal spasm. This is due to a tight contraction of the vocal cords and is recognized by a high pitched sound during inspiration. The condition will usually resolve by delivering 100% oxygen by mask and waiting for further inspiratory effort. If laryngeal spasm occurs, the diagnosis of acute respiratory failure is probably incorrect.

Obstruction of the airway below the larynx is less common but may arise from excessive bronchial secretions, mucosal oedema, due to irritants such as smoke bronchospasm, (as in asthma) and aspiration of gastric contents.

### 5.2.1.2 Recognition of Airway Obstruction

Recognition of airway obstruction may be difficult in cases of partial respiratory failure. The classical ‘look, listen and feel’ approach discussed in the previous chapter is a simple, systematic method of detecting airway obstruction, both in primary and secondary assessments of breathing.

*Primary (flash) examination*

For a rapid assessment of breathing:

1. *Look* for the presence of chest and abdominal movements.
2. *Listen* and *feel* for airflow at the mouth and nose.

In partial airway obstruction, air entry is diminished and usually noisy. Inspiratory stridor is caused by obstruction at the laryngeal level or above. There are characteristic sounds heard at the mouth associated with airway obstruction.

*Expiratory wheeze*: Implies obstruction of the lower airways, which tend to collapse and obstruct during expiration.

*Gurgling*: Indicates the presence of liquid or semi-solid foreign material in the large airways.

*Snoring*: arises when the pharynx is partially occluded by the soft palate or epiglottis.

*Crowing* is the characteristic sound of laryngeal spasm.

*Secondary examination*

*Look*

In a patient who is making respiratory efforts, complete airway obstruction causes paradoxical chest and abdominal movement, often described as ‘see-saw’ breathing. As the patient attempts to breathe in, the chest is drawn in and the abdomen expands; the opposite occurs during expiration. This is in contrast to the normal breathing pattern of synchronous movement upwards and outwards of the abdomen (pushed down by the diaphragm) with the lifting of the chest wall. During airway obstruction, other accessory muscles of respiration are used, with the neck and the shoulder muscles contracting to assist movement of the thoracic cage.

*Listen*

This supplements listening at the in the primary assessment and requires skill in using a stethoscope. This can only come from training and experience in using the device rather than simply wearing it around the neck as a badge of office, as has become fashionable in recent years. Full examination of the neck, chest and abdomen is required. The examination must include listening for the absence of breath sounds in order to diagnose complete airway obstruction reliably; any noisy breathing indicates partial airway obstruction.

During apnoea, when spontaneous breathing movements are absent, complete airway obstruction is recognised by failure to inflate the lungs during attempted positive pressure ventilation.

## 5.3 Basic Airway Management

Once partial or complete airway obstruction is recognised, immediate measures must be taken to create and maintain a clear airway. An immediate response is to place the patient in the left lateral position to reduce obstruction by the tongue falling back. Once care is available the patient can be moved to the supine position for further care. There are three manual manoeuvres that may improve the patency of an airway obstructed by the tongue or other upper airway structures without the use of airway adjuncts. These are particularly useful when no equipment is immediately to hand as is often the case in basic life support. These manoeuvres are: (1) head tilt, (2) chin lift, and (3) jaw thrust.

### 5.3.1 Head Tilt and Chin Lift

In this manoeuvre the rescuer's hand is placed on the patient's forehead and the head gently tilted back; the fingertips of the other hand are placed under the point of the patient's chin, which is lifted gently (see Fig. 5.2).

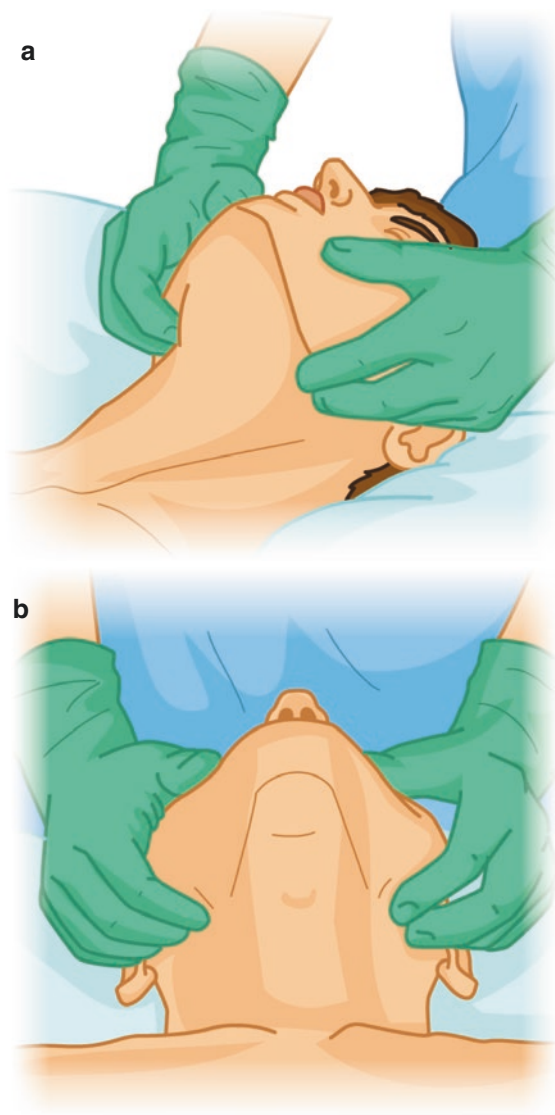
**Fig. 5.2** Head tilt and chin lift



### 5.3.2 *Jaw Thrust*

Jaw thrust is an alternative method of bringing the mandible forward and relieving obstruction by the soft palate and epiglottis. The rescuer's index and other fingers are placed behind the angle of the mandible, and pressure is applied upwards and forwards. Using the thumbs, the mouth is opened slightly by downward displacement of the chin (Fig. 5.3).

**Fig. 5.3** Jaw thrust



These simple positional methods can be applied quickly and are effective in most cases where airway obstruction results from relaxation of the soft tissues.

If a clear airway cannot be achieved this way look for other causes of obstruction such as a foreign body. These can be detected and removed with a finger sweep inside the mouth or using forceps if available. Broken or displaced dentures should be removed, but well-fitting dentures should be left in place as they help to maintain the contours of the mouth, facilitating a good seal for ventilation by mask.

### ***5.3.3 Airway Management in Patients with Suspected Cervical Spine Injury***

In the case of possible trauma to the neck excessive head tilt could aggravate the injury and damage the cervical spinal cord. In such a case establish a clear upper airway by using jaw thrust or chin lift in combination with manual in-line stabilisation (MILS) of the head and neck by an assistant. If life-threatening airway obstruction persists despite effective application of jaw thrust or chin lift, the head may be tilted in small increments until the airway is open. Remember that establishing a patent airway takes priority over concerns about a potential cervical spine injury although manoeuvres to protect the airway must be integrated with the management of the traumatic injury.

### ***5.3.4 Aids to Positional Airway Management Techniques***

The methods of early control of the airway described above can be supplemented by the use of nasal and oropharyngeal airways.

The ILCOR guidelines note that despite a lack of published data on the use of nasopharyngeal and oropharyngeal airways during CPR, they are often helpful, and sometimes essential, to maintain an open airway, particularly when resuscitation is prolonged. Many of the established techniques in airway and ventilation lack a modern clinical evidence base. The use of suction to clear secretions, used universally is a case in point.

#### **5.3.4.1 Suction**

The use of suction (aspiration) in clearing the upper airway is essential and is an often forgotten part of resuscitation training. It is important to use a suction tube that is the widest bore possible to be able to deal with viscid secretions and vomitus that may contain food particles. The author has always tested the performance of any

**Fig. 5.4** The Vitellograph pistol grip suction device. This is equipped with a wide bore tube which is effective in clearing the upper airway secretions quickly (Reproduced with permission: Vitellograph, UK)



sucker using condensed soup as a test material. Do not use gas powered suckers; these are often powered by compressed oxygen and waste gas unnecessarily. The best suction devices to use in emergency are of the pistol grip type such as the Vitellograph. These remove the secretions through a wide bore suction tube into a chamber contained in the device (Fig. 5.4). When a patient has been intubated a narrower bore catheter will be necessary to clear any secretions from the ETT.

It is important to use the sucker carefully if the patient is only lightly unconscious and has an intact gag reflex since pharyngeal stimulation can provoke vomiting and possible laryngeal spasm.

#### 5.3.4.2 Oropharyngeal Airways

Oropharyngeal (Guedel) airways help prevent the tongue from obstructing the pharynx. Guedel airways are available in sizes suitable for the newborn to large adults. An estimate of the size required is obtained by selecting an airway with a length corresponding to the vertical distance between the patient's incisors and the angle of the jaw.

The most common sizes are 2, 3 and 4 for small, medium and large adults, respectively. Note that if the patient is partially conscious and reflexes are still present insertion of an oropharyngeal airway may cause vomiting or laryngospasm. Therefore insertion should be attempted only in comatose patients. Figure 5.5 shows the correct way of inserting a Guedel airway. The device should be inserted with the curve facing upwards and then turned in the mouth so that the curve rests over the tongue.

#### 5.3.4.3 Nasopharyngeal Airways

The potential problems in using an oropharyngeal airway in a patient who is partially conscious can be overcome using a nasopharyngeal airway which is better tolerated. The nasopharyngeal airway is potentially life-saving when insertion of an oral airway is impossible. The traditional methods of sizing a nasopharyngeal

**Fig. 5.5** Inserting an oropharyngeal (Guedel) airway. The device should be inserted with the curve upwards and then turned in the mouth to cover the tongue



airway (measurement against the patient's little finger or anterior nares) do not correlate with the airway anatomy and are unreliable. Sizes of 6–7 mm are suitable for adults. Insertion can cause damage to the mucosal lining of the nasal airway, resulting in bleeding in up to 30% of cases. *Caution:* If the tube is too long it may stimulate the laryngeal or glossopharyngeal reflexes to produce laryngospasm or vomiting.

## 5.4 Advanced Airway Management

### 5.4.1 Introduction

As we have seen above artificial ventilation can be delivered using basic airway management techniques through a pharyngeal mask connected to a bag valve device or portable ventilator. However, as has been discussed previously the main problems associated with AV via an unprotected airway are Inflation of the stomach and regurgitation and inhalation of stomach contents. Therefore when AV is being used a secure airway should be established as soon as possible. The success of doing so however will depend on a number of factors, such as the skills and experience of the operators and anatomical difficulties in the patient. Advanced airway management is a clinical skill that must be taught and practised, both on manikins and in the controlled circumstances of the anaesthetic room in the operating unit. No book description can ever replace this training. The following notes, based upon



recommendations of the UK Ambulance staff training manual are provided to act as an aide memoire for advanced airway management by emergency paramedical and medical staff. Under no circumstances should advanced airway management be attempted without proper training.

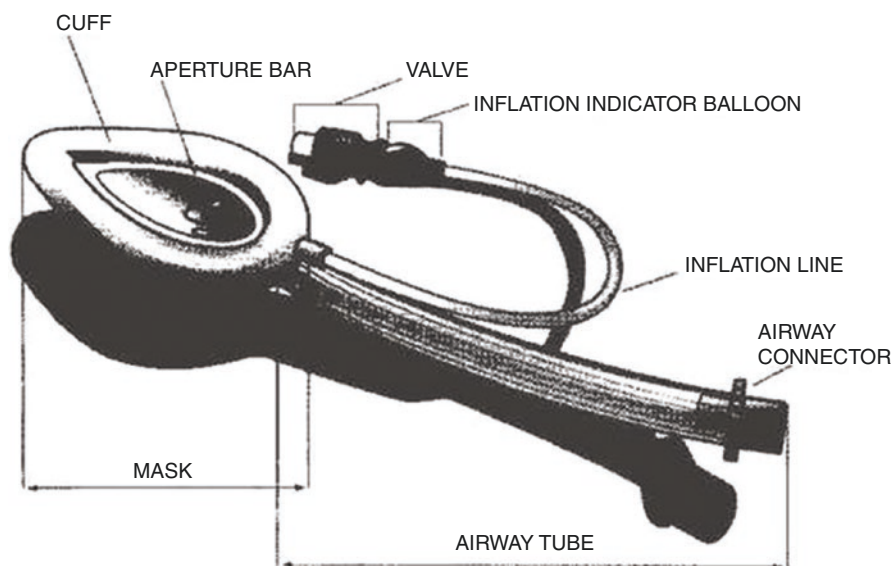
### **5.4.2 Which Airway to Use?**

For many years intubation of the trachea has been regarded as the definitive way of providing a secure airway for AV. This is essentially true in hospital settings where intubation is being done by anaesthetists, emergency and intensive care physicians who perform it regularly and are well-experienced in dealing with potential difficulties. The situation for non-specialist providers of AV is substantially different. Although good training in intubation will have been provided, the number of intubations required to be performed each year is often way below that which would ensure retention of intubation skills. In life-threatening hypoxia therefore valuable time may be wasted in repeated attempts to insert an ETT when other techniques would have been more appropriate. The ETT is only the 'gold standard' of airway management if it can be inserted. The chief alternative to intubation which has been developed over the past 30 years is the laryngeal mask airway (LMA), developed by Dr. Archie Brain in the UK in the early 1980s. This should probably be regarded now as the primary method of choice for establishing a protected airway for those who are less experienced in intubation. Each different emergency care service will have its own protocols for airway management but there is increasing consensus that the LMA should be the first choice.

### **5.4.3 The Laryngeal Mask Airway**

The LMA (Fig. 5.6) is composed of a wide-bore plastic tube with an inflatable mask-like cuff at its further end. This cuff is designed to sit above the larynx in the same way the pharyngeal mask sits over the mouth. The advantage of the LMA is that it provides a clear and protected airway which can be inserted without the use of a laryngoscope or training in intubation. It has been shown to be suitable for AV provided that the inflation pressure is not too high (20 cm H<sub>2</sub>O). Although it does not provide the same degree of airway protection at higher pressures as the ETT the relative ease and speed of insertion make it very valuable in emergency ventilation.

Box 5.1 describes the steps in insertion of the LMA. Before attempting insertion ensure that the patient has received several breaths of 100% oxygen via basic airway and ventilation management.



**Fig. 5.6** The components of the laryngeal mask airway

### **Box 5.1 Insertion of the LMA: The Essential Steps**

1. Select the correct size of mask to be used

- Size 3: child/small adult
- Size 4 normal adult
- Size 5 large adult

In addition to the mask, other equipment required includes a 20 ml syringe or bigger, fixing tape, a pistol grip sucker and lubricating gel.

2. Ensure that the mask is deflated as much as possible by attaching to the syringe and withdrawing any remaining air. If there is a loss of vacuum the LMA should be changed before use. Always deflate the mask so that the rim faces away from the mask aperture. There should be no folds near the tip. Before use the LMA must be thoroughly lubricated
3. Gently suck out any copious secretions or vomitus from the mouth taking care not to insert the suction tube back to the pharynx which might cause laryngeal stimulation and spasm. Under direct vision, insert the mask, pressing upwards against the hard palate to flatten it out. Use the index finger to keep pressing upwards as the mask is inserted into the pharynx to ensure that the tip remains flattened and avoids the tongue
4. With the neck flexed and the head extended press the mask into the posterior pharyngeal wall using the index finger
5. Continue pushing with the tip of the index finger to guide the mask down to its final position over the larynx. If the other fingers are kept clear and

the forearm is slightly rotated inwards the mask may be placed in position in one complete movement

6. Next, grasp the tube with the other hand and withdraw the guiding index finger from the pharynx. Press downwards on the mask with the other hand to ensure that the mask is fully inserted
7. Inflate the mask with the recommended volume of air. Do not overinflate
  - Size 3: up to 20 ml
  - Size 4: up to 30 ml
  - Size 5: up to 40 ml
8. It is important not to touch the LM tube while inflating unless the position is obviously unstable, as may be the case in older patients with slack tissues. With inflation the mask will rise slightly out of the hypopharynx, seating itself in the correct position. When the mask is in place, insert a suitable Guedel airway alongside it to provide protection against the patient biting on the tube of the LMA and causing obstruction. Secure the LMA with a tied tape or adhesive tape
9. The mask may now be connected to bag-valve device or portable ventilator and ventilation started. The corrected placement of the LMA will be confirmed with observation of the chest rise and auscultation of air entry into the right and left lungs. There should be no audible leak of air from the mouth around the LMA tube which would indicate an under-inflated mask

#### 5.4.3.1 Potential Problems with Insertion and Function of the LMA

Problems during insertion

1. Inadequate neck flexion. If the LMA will not advance, withdraw it and re-insert with the neck fully flexed. The usual caution must be applied if there is a possibility of cervical spine injury.
2. Incorrect or incomplete deflation before use or inadequate lubrication. Remove the device and re-insert.
3. Incorrect insertion technique. Revert to ventilation using an unprotected airway if necessary.
4. Presence of an anatomical or pathological obstruction. Attempt reinsertion or revert to unprotected airway ventilation with a pharyngeal mask if necessary.

Problems with apparent obstruction or malfunction of the LMA

1. Rotation to the lateral or posterior position. Reposition so that the indicated line on the tube faces upwards.
2. Insertion too deeply. Gently withdraw the LMA until a satisfactory airway is obtained.
3. The epiglottis has become folded down. Remove and re-insert the LMA.

4. Wrong size LMA. Remove and re-insert another size.
5. Incorrectly seated or folded over; Remove and re-insert the LMA.
6. There is an inadequate cuff volume. Check the pressure in the pilot balloon.
7. The LMA cuff is punctured (always check the cuff during routine equipment checks before attending an emergency).
8. Low pulmonary compliance and high airway resistance. These may be caused by pulmonary oedema and bronchospasm respectively.
9. Patient biting on the LMA tube. Always insert a Guedel airway alongside the LMA as a 'bite block' to prevent occlusion of the tube in this way.

#### Regurgitation during use

If fluid appears in the airway during use do not deflate or remove the device. Fluid may arise from either the stomach or the lungs. Suction should be performed thoroughly in the tube of the LMA and in the pharynx.

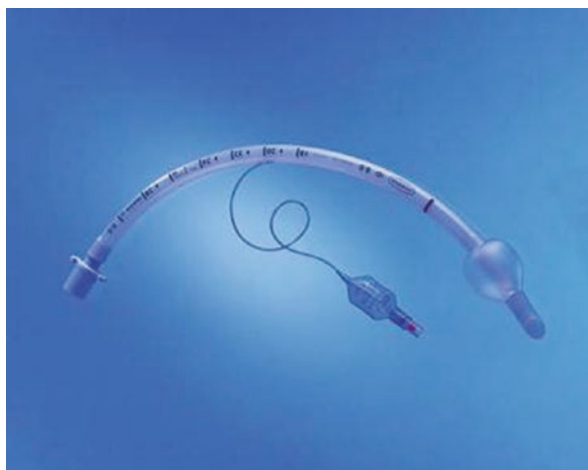
### 5.4.3.2 Contraindications to the Use of an LMA

The relative contraindications for the use of the LMA in AV would be conditions where high inflation pressures are likely (chronic obstructive airways disease, asthma). Other contraindications include a full stomach or severe facial trauma.

### 5.4.4 Endotracheal Intubation

A modern disposable ETT is shown in Fig. 5.7. As mentioned above, the following notes are provided as an aide memoire for its use. They do not replace the essential requirement for training in intubation.

**Fig. 5.7** A typical disposable plastic adult endotracheal tube (Photograph courtesy of Smiths Medical International, Luton, United Kingdom)



#### 5.4.4.1 Standard Equipment Checks

Individual emergency services provide standard equipment checks for all emergency equipment before the emergency arises (for example at the start of an ambulance shift). These checks are important for the equipment to be used for intubation. A suggested check list is provided in Box 5.2.

##### **Box 5.2 Intubation Equipment Checklist**

All intubation equipment should be checked on a regular basis according to the procedures of the service within which the operator is working. A basic checklist should cover the following:

1. Laryngoscope

- Do the blade and handle fit correctly?
- Is the blade the correct size?
- Do the bulb and battery work correctly. Are spares present?
- Is the bulb secure?

2. Endotracheal tubes

- Is there a selection of tube sizes cut to the correct length?
- Does each tube have an airtight cuff and secure connectors?

3. Catheter mount

Is the catheter mount intact and undamaged? Does it swivel through 360 deg?

4. Suction apparatus

- Is a suitable pistol grip sucker available? Does it work correctly?
- Are suction catheters available which fit the size of ETT used?

5. Other items: check that these are present

- Lubricating jelly
- Magill forceps
- Stethoscope
- Intubating stylet
- Oropharyngeal airways
- Syringe for inflation of the pilot balloon
- Ribbon gauze/tape
- Spencer Wells forceps

##### **Endotracheal tube preparation**

Almost all ETT nowadays are disposable and made of plastic. These are packaged uncut and too long for intubation. If an ETT is too long there is a

risk of intubating the right main bronchus. For emergency use the tubes should be pre-cut to the correct length given by the following table:

Age	Internal diameter (mm)	Length (cm)
6 months	3.5–4.0	9.5
1 year	4.0	12.5
2 years	4.5	13
4 years	5.0	14
6 years	5.5	15
8 years	6.0	16
10 years	6.5	17
12 years	7.0	18
14 years	8.0	19
Adult male/female	8.0–9.0	22–24

When cut the tubes should be returned to their original packaging, clearly labelled and with the connectors firmly inserted in place.

In some services, paediatric tubes are left uncut and the extra length used in place of a catheter mount after intubation to reduce dead space.

The table above calculates the internal diameter and length of paediatric tubes according to the following formulae.

$\text{Internal diameter (mm)} = \text{age in years} / 4 + 4$

$\text{Tube length (for infants up to puberty)} = \text{age in years} / 2 + 12$

**5.4.4.2    Adult Intubation**

Before intubation the patient should be ventilated with a few breaths of 100% oxygen from a mask to achieve the highest possible concentration during the period of intubation so that the patient does not become hypoxic. Remember that during intubation the patient is not being ventilated if total respiratory failure is present. Therefore the time taken for intubation should be as short as possible. One tip is to hold your breath during intubation. If you need to breathe so does the patient. If intubation has not been achieved by the time you need to breathe abort the attempt and reventilate the patient by mask. Box 5.3 outlines the steps of endotracheal intubation. Remember that hands-on training is an essential first step before using this aide memoire.

**Box 5.3 Steps in Endotracheal Intubation**

1. Check all equipment while the patient is still being ventilated via a basic airway. Pre-intubation equipment checks should cover
  - Availability
  - Function
  - Compatibility
  - Correct tube size, cuff function and suction
2. Pre-oxygenate the patient using bag–valve device with supplemental oxygen
3. Position the patient
  - Always consider the possibility of cervical spine injury
  - If this is the case use, manual in-line stabilisation to bring the upper airway in line with the trachea
  - Place a small towel or pillow under the patient's head to help achieve the optimal intubating position with the neck forward and head backward (sometime described as 'sniffing the morning breeze').
4. Open the patient's mouth
  - Check for loose teeth and foreign bodies and remove
  - Clear all secretions with the sucker and re-oxygenate with the BVM
5. Laryngoscopy
  - Remember that all actions should be as gentle as possible
  - Insert the laryngoscope into the right side of the mouth, displacing the tongue to the left
  - Advance the tip of the laryngoscope backwards into the pharynx until the tip of the epiglottis is seen
  - Position the tip of the blade in front of the epiglottis and gently lift the laryngoscope upwards to reveal the larynx and vocal cords. Never lever the laryngoscope on the teeth
6. Insert the tube under direct vision
  - Make sure you have correctly identified the vocal cords and laryngeal opening
  - Once this is verified insert the lubricated tube under direct vision between them into the trachea
  - Be careful to pass only enough tube so that the cuff is positioned just below the cords

### 7. Inflate the cuff

Connect the syringe to the cuff and inject 7–10 ml air to seal the tube in place and avoid any further risk of the inhalation of vomitus, secretions or debris

### 8. Connect the catheter mount and bag valve mask

Connect the bag valve mask via the catheter mount and ventilate the patient manually. If air is heard leaking around the cuff inflate it further until the sounds cease

### 9. Ventilate and check the position of the tube

Ventilation can now be continued using the BVM or a portable ventilator. It is important to check the position of the tube as follows:

Check for symmetrical chest movements (note that there may be abdominal movements also)

Use a stethoscope to check that air is entering the lungs equally on both sides (listen over the mid-axillary line on both sides)

Beware of intubation of the right main bronchus which can be detected by breath sounds being heard on the right but not the left side. If this occurs, deflate the cuff briefly and withdraw it slightly. Re-inflate the cuff and check that the breath sounds are now equal on both sides

If breath sounds are not heard and there is no chest movement the tube may have entered the oesophagus. This is indicated by gurgling sounds over the stomach (below the margin of the left ribs). If this is the case withdraw the tube, re-oxygenate the patient by mask and recommence the intubation. It may be wise to hand over to another operator at this stage.

### 10. Secure the tube

After checking the position of the tube and correct air entry it must be securely fastened with tied tape or adhesive plaster. Insert an oropharyngeal airway alongside the tube to protect it against the patient biting and blocking the tube if there is an improvement in consciousness.

After securing the tube check the position and air entry again to make sure that it has not become displaced during securing. Note that after any movement of an intubated patient the position of the ETT should be checked again.



### 5.4.4.3 Overcoming Difficulties During Intubation

There may be a number of unforeseen difficulties during intubation which can affect all operators, even the most experienced anaesthetists. Always remember to call for help if problems arise.

Potential problems:

1. There is always a small risk of incorrect tube placement during intubation. This is increased in some patients by difficulties in visualising the vocal cords

There may be only a partial view or no view at all. The following actions may help:

- Head and neck position
  - Check that the head position described above is being used.
  - Try applying external pressure by a colleague backwards to the larynx.

If these measures do not improve the view revert immediately to BVM ventilation and insert a laryngeal mask after re-oxygenation. If this is not possible, continue ventilation with an unsecured airway until further expert help is available.

In some cases a clear view of the cords can be obtained but the end of the ETT cannot be made to align with the laryngeal inlet. In this situation, withdraw the tube and laryngoscope from the mouth and continue ventilation by mask. Ask an assistant to introduce an intubating stylet into the ETT. When intubation is then re-attempted it will be found that the tube can be bent to shape to enter the laryngeal opening and trachea.

If the tube cannot be passed through the cords do not use force but consider repeating the intubation using a smaller diameter tube.

Paediatric intubation is usually conducted by specialists. The main stages are considered in Chap. 10.

## 5.5 Oxygen Therapy for Partial Respiratory Failure

After the airway has been established, if there is partial respiratory failure the patient is still breathing but either has poor breathing efforts (for example as a result of decreased conscious level or trauma). The result of the partial respiratory failure is the build-up of carbon dioxide due to type 2 respiratory failure. As the alveolar air equation (Chap. 3) shows this will lead to a reduced partial pressure of oxygen in the alveoli when breathing air with an  $\text{FiO}_2$  of 0.21. However if the  $\text{FiO}_2$  is increased the alveolar oxygen level will rise. This is the basis for the use of free flow oxygen through a face mask in cases of partial respiratory depression. In emergency a concentration of 100% is used but this should be reduced as soon as possible since there

are established dangers from breathing this concentration for too long... When the emergency is stabilized and the patient has improved oxygen saturation and blood oxygen levels the inspired concentration is usually reduced to 40%. Free flow oxygen is usually delivered through a light face mask at a rate of 6–8 litres/minute set by the regulator valve on the cylinder. This is a wasteful use of oxygen and can be better replaced by the use of a demand delivery system found on many portable ventilators (Chap. 7). In situations where mass respiratory support is required there is a major requirement to conserve oxygen which may be in short supply. The COVID 19 pandemic has brought this point home forcefully and there is now greater awareness of how precious a resource therapeutic oxygen is.

One important point about free flow oxygen in partial respiratory failure is that it must be used with caution in a patient who has a history of chronic obstructive airways disease (chronic bronchitis). These patients become adapted to high levels of carbon dioxide which normally would promote breathing via receptors in the medulla of the brain. These receptors no longer control breathing and instead oxygen detector receptors situated in the aorta control breathing by detection of low oxygen levels in the blood. If these levels suddenly rise as a result of oxygen therapy the hypoxic drive mechanism will fail and the patient may stop breathing. Therefore when treating partial respiratory failure in an acute or chronic episode of bronchitis the respiratory depth and rate should be monitored carefully after starting oxygen therapy.

## **5.6 Techniques for Non-mechanical Methods of Artificial Ventilation**

Artificial ventilation is required when the patient's breathing efforts are inadequate or absent. Here we consider the manual (non-mechanical) methods of providing subsequent artificial ventilation. Mechanical methods of AV are the subject of the next chapter.

### ***5.6.1 Expired Air Ventilation***

Having cleared and established the airway it is essential to provide artificial ventilation as soon as possible for any patient in whom spontaneous ventilation is inadequate or absent. Ventilation by mouth to mouth or mouth to nose was a basic part of the original CPR technique developed in the late 1950s by Elam and Safar. It has the advantage of being easily taught and requires no equipment. Recently there has been increasing resistance from the general public to use this technique largely from

fear of contagion which has led to the adoption of compression only CPR for cardiac arrest. In the case of primary respiratory arrest however expired air ventilation remains potentially life-saving.

Expired air ventilation (rescue breathing) is effective since the rescuer's expired oxygen concentration is 16–17%, which still provides a saturation of about 90% (see Chap. 3) due to the sigmoidal shape of the haemoglobin dissociation curve.

### 5.6.1.1 Delivering Mouth to Mouth Ventilation

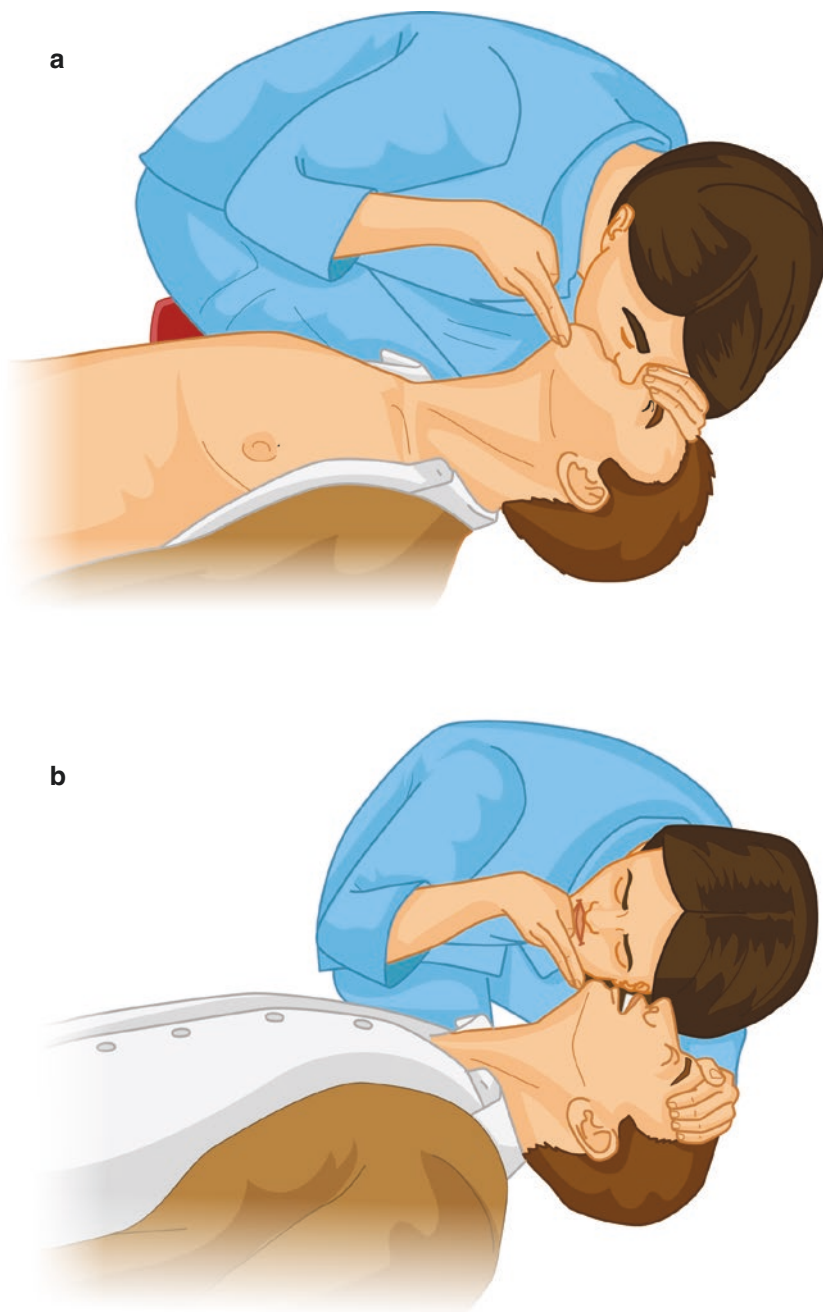
Expired air ventilation is used in basic cardiopulmonary resuscitation and the ILCOR guidelines on mouth to mouth ventilation are as follows:

1. The basis of rescue CPR is to deliver two mouth to mouth ventilations followed by 30 chest compressions at a rate of 100/min
2. Low minute-ventilation (lower than normal tidal volume and respiratory rate) can maintain effective oxygenation and ventilation. During adult CPR, tidal volumes of approximately 500–600 ml (6–7 ml kg<sup>-1</sup>) are recommended. The delivery of an effective tidal volume has to be estimated by eye by looking at the chest movement
3. Rescuers should give each rescue breath over about 1s, with enough volume to make the patient's chest rise, but to avoid rapid or forceful breaths. The time taken to give two breaths should not exceed 5s. These recommendations apply to all forms of ventilation during CPR, including mouth-to-mouth and bag-mask ventilation with and without supplementary oxygen.
4. Mouth-to-nose ventilation is an acceptable alternative to mouth-to-mouth ventilation. It may be considered if the mouth is seriously injured or cannot be opened or a mouth-to-mouth seal is difficult to achieve (Fig. 5.8)

### 5.6.1.2 The Pocket Mask

A more socially-acceptable alternative to mouth to mouth ventilation is the pocket resuscitation mask shown in Fig. 5.9. It is similar to a pharyngeal mask and allows expired air ventilation without direct contact. It has a unidirectional valve, which directs the patient's expired air away from the rescuer. The mask is transparent so that vomit or blood from the patient can be seen. Some masks have a connector for the addition of oxygen. When using masks without a connector, supplemental oxygen can be given by placing the tubing underneath one side and ensuring an adequate seal.

Using the pocket mask for expired air ventilation, a two-hand technique should be used to maximize the seal with the patient's face. The timing of the breaths should be the same as for basic mouth to mouth ventilation.



**Fig. 5.8** Expired air (rescue) ventilation. (a) Blow steadily into the mouth and watch the chest rise (b) watch the chest fall during expiration, keeping the airway open

**Fig. 5.9** Using a pocket mask for expired air ventilation. Note the two handed grip to ensure a good seal of the mask around the face



### 5.6.2 *The Bag–Valve Mask Device*

The bag valve mask (BVM), is an oval-shaped hand-held device commonly used to provide manual positive pressure artificial ventilation. The device is a required part of resuscitation equipment for trained professionals in out-of-hospital settings (such as ambulance crews) and is also frequently used in hospitals as part of standard equipment found in anaesthetic rooms, on emergency resuscitation trolleys, in emergency rooms and in intensive care units. Emphasizing the frequency and prominence of BVM use in the United States, the American Heart Association (AHA) Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiac Care recommends that all healthcare providers should be familiar with the use of the bag-mask device. The use of the device for primary resuscitation is recommended by the ILCOR guidelines.

#### 5.6.2.1 **Development**

The bag-valve mask concept was developed in 1953 by the German engineer Holger Hesse and his partner, Danish anaesthetist Henning Ruben. They named their resuscitator the Ambu bag, (Artificial Manual Breathing Unit) and then formed their own company, also called Ambu, to manufacture and market the device beginning in 1956. As the first brand of manual resuscitator to go to market, led to the name Ambu becoming a generic trademark, with resuscitators from any manufacturer commonly being referred to as ‘Ambu bags’. A commercial comparison is the use

**Fig. 5.10** An AMBU self reforming bag (Photograph courtesy of AMBU Copenhagen)



of the term 'Hoover' as an alternative name for a vacuum cleaner. Henning Ruben also developed a non-return valve to ensure that expired carbon dioxide did not mix with the delivered air and oxygen from the bag. This was an essential feature of the final version of the AMBU bag (Fig. 5.10).

### 5.6.2.2 Construction of the Bag-Valve-Mask

The BVM consists of a flexible oval shaped bag air chamber attached to a face mask (pharyngeal mask) via a non-return (Ruben) valve. The shape arose from the Boyle reservoir bag used on anaesthetic machines. This was used before the introduction of mechanical ventilation in the operating theatre to provide assisted ventilation and was kept open by the pressure of gas from the cylinders of the anaesthetic machine. Ruben contrived to use springs (made from metal coat hangers) to keep the bag open without pressurized gas inside (hence the term 'self reforming bag'). The original version with springs was replaced with foam interior to ensure reforming.

Many modern bags used in resuscitation outside the hospital are now made of disposable materials, usually silicone rubber. The composition of the material used to make the bags has the property of being self-reforming so that when the bag is squeezed it returns to its original shape and draws in air from the outside through the entry port. For bags that are to be used in toxic environments butyl rubber is used. This material has the advantage of being resistant to the passage of toxic chemicals (Figs. 5.11 and 5.12).

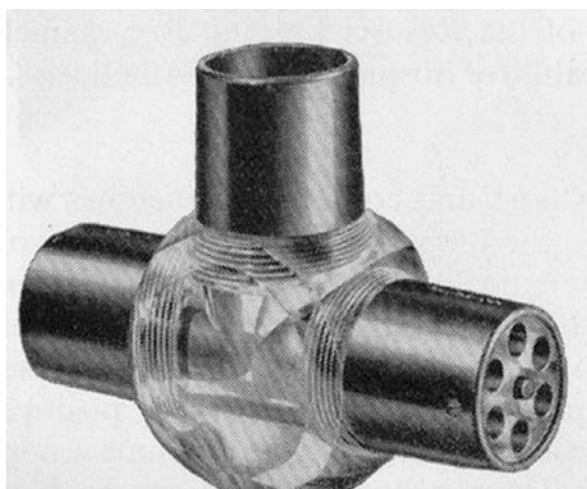
### 5.6.2.3 Non-return Valve and pharyngeal mask

The bag is connected to the mask by a non-return valve (also known as a Ruben valve). This valve ensures that the carbon dioxide breathed out from the patient does not mix with the air or oxygen being delivered by squeezing the bag. The non-return



**Fig. 5.11** The AMBU Mk 3 butyl variant bag valve device. Resuscitation is often required in a zone contaminated by a chemical agent. In this situation AV is provided by responders dressed in protective suits. A special version of the BVM (the AMBU butyl variant) can be used in this situation. It is constructed from butyl rubber which does not allow penetration by chemical agents. In this device the ambient air used to ventilate the patient is filtered through a canister, see on the right of the picture, containing activated charcoal which removes the chemical pollution. It should be noted that this device with a filter should not be used in an environment where there is a reduced oxygen concentration as in the case of many fires. Also the filter does not remove carbon monoxide from the inhaled atmosphere. (Author's image)

**Fig. 5.12** The Ruben non-return valve



valve connects to a mask, known as a pharyngeal mask which fits around the mouth of the patient. Original masks were made of rigid black rubber but modern versions have an inflated perimeter (known as 'soft seal') which allow an airtight seal with the patient without having to use undue pressure (Fig. 5.10 (1)).



#### **5.6.2.4 Oxygen Enrichment**

In basic use the BV device ventilates the patient using air which is drawn in at the rear end of the bag via one way entry valve. To produce higher concentrations of oxygen during ventilation the bag is fitted with a connection to allow the entry of free flow oxygen at atmospheric pressure. The higher the flow of added oxygen the greater will be the concentration in the mask. In theory, a concentration of 100% should be possible with an oxygen flow equivalent to the delivered minute volume. However in practice this is rarely the case due to leaks around the mask.

### **5.6.3 Using the Bag Valve Device**

When the face mask is properly applied and the bag is squeezed the device forces air through into the patient's lungs; when the bag is released, it self-inflates from its other end, drawing in either ambient air or a low pressure oxygen flow supplied by a regulated cylinder, while also allowing the patient's lungs to deflate to the ambient environment (not the bag) past the one way valve.

#### **5.6.3.1 How Much and How Fast to Squeeze the Bag?**

In order to be effective, a bag valve mask must deliver between 500 and 800 ml of air to a normal male adult patient's lungs with each ventilation but if supplemental oxygen is provided 400 ml may still be adequate. However the delivered tidal volume cannot be measured using a BVM so the effectiveness of delivering a tidal volume depends on observing the rise and fall of the chest. Compare what is being delivered with the chest movements during normal respiration. Squeezing and releasing the bag once every 5–6s for an adult or once every 3s for an infant or child provides an adequate respiratory rate (10–12 respirations per minute in an adult and 20 per minute in a child or infant).

In practice, when using the bag it is important to squeeze it gently to about half its original inflated volume. It is essential not to squeeze too hard or too fast, otherwise hyperinflation will occur. A rate of 12 ventilations per minute is recommended. However, in emergency it is very easy to exceed this rate. Practice with a musical metronome is useful in training. With the metronome set to 36 beats per minute, inspiration should take one beat while expiration should take two beats. This gives a correct I:E ratio of 1:2.

The bag and valve can also be attached to an alternate airway adjunct, instead of to the mask, for example an endotracheal tube or LMA.



### 5.6.3.2 One and Two Person Ventilation with the Bag–Valve–Mask

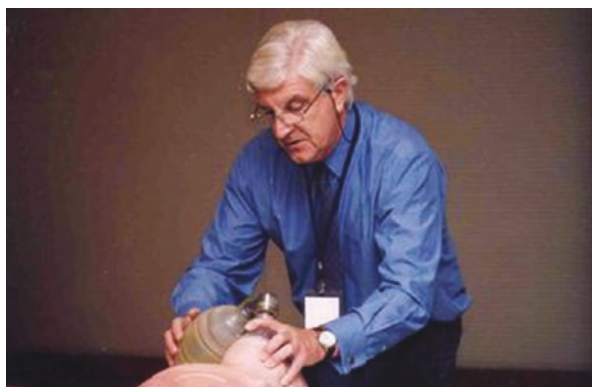
Although the bag–valve—mask device enables ventilation with high concentrations of oxygen, its use by a single person requires considerable skill. The correct position of the hand in holding the mask is shown in Fig. 5.13. The thumb and forefinger are placed around the upper part of the mask while the remaining fingers are placed under the mandible. Skilled users of this position will also be able to palpate the facial artery with the three fingers as it passes under the jaw. When being used by one operator is often difficult to achieve a gas-tight seal between the mask and the patient's face, and to maintain a patent airway with one hand while squeezing the bag with the other. Any significant leak will cause hypoventilation and, if the airway is not patent, gas may be forced into the stomach. This will reduce ventilation further and greatly increase the risk of regurgitation and aspiration.

For this reason the two-person technique for bag-mask ventilation is preferable (Fig. 5.14). One person holds the facemask in place using a jaw thrust with both hands, (the double C position) while an assistant squeezes the bag. In this way, a better seal can be achieved and the patient's lungs can be ventilated more effectively and safely. The first member of the team focuses on maintaining a good seal around the mask and keeping the airway open while the second focuses on delivered breaths and timing, while watching the chest.

### 5.6.4 Perceptions of the Safety and Effectiveness of Bag Valve Mask Ventilation

Since its invention the BVM device has been adopted for use world-wide by both medical and paramedical personnel. In many areas, particularly the United States it is the only authorised method of providing emergency ventilation.

**Fig. 5.13** Single handed ventilation using the BVM device (Author's image)





**Fig. 5.14** Two person ventilation using the BVM. The operator holding the mask has the hands on the 'double C' position, which ensures a good seal of the mask while also allowing head-tilt and chin lift to keep the airway open

The reasons for the popularity of the device may be summarised as follows:

1. It had an apparent simplicity of action with one hand holding the mask while the other squeezed the bag (single operator) or two hands holding the mask (the so-called 'double C' position) and another person squeezing the bag.
2. There was a belief that in squeezing the bag manually there was a feeling of being in 'direct contact' with the patient's lungs and therefore over-ventilation would be avoided.
3. There was a conviction that the device was essentially safe to use and could provide effective ventilation. This was probably based upon the fact that that bag

ventilation is used following the induction of general anaesthesia in the anaesthetic room on patients who are asleep, have muscle relaxation and usually an empty stomach. Many of these patients would also have normal values of airway resistance and lung compliance. BVM ventilation in these circumstances is relatively straightforward. It is in this setting that many paramedics first receive their airway and ventilation training and the experience with the BVM is carried away into the ambulance services.

4. Compared with mechanical ventilators providing BVM involved a low financial outlay. The development of disposable BVM at the end of the twentieth century provided a solution to sterilisation with increasing concerns about cross-infection.

### ***5.6.5 Problems Associated with the Use of the Bag-Valve—Mask Device***

Despite continuing widespread use of the BVM in emergency there has been increasing concern over the past decade about potential serious problems that may be associated with this type of positive pressure ventilation. These are related to both hypo- and hyper-ventilation.

#### **5.6.5.1 Gas Wastage and Hypoventilation**

The BVM usually operates using air as the main gas with supplemental free-flow oxygen provided to increase the oxygen concentration. However this can cause considerable wastage of bottled oxygen due to leaks around the mask (particularly if held with only one hand). In addition, if the mask seal is not effective too low a tidal volume will be delivered to the patient leading to hypoventilation, with a low minute volume and hypoxia.

#### **5.6.5.2 Hyperventilation**

Inappropriate use of the BVM may cause (1) high ventilation frequency and tidal and minute volumes and (2) excessive inflation pressures. These can cause gastric insufflation and barotrauma, where weak sections of the lung tissue are disrupted, leading to pneumothorax. There is also the more - recently recognised problem of volutrauma, where damage is caused to the alveoli by over-distension (see Chap. 11).

### High ventilation rates

Ventilating the patient too quickly, even if the tidal volume is correct leads to an excessive minute volume and hypocapnia. Even with trained and experienced operators the stressful nature of the emergency situations where BVM are used can lead to high ventilation rates. Several studies have confirmed the problem of high ventilation rates. Cooper and colleagues (2006) found that keeping artificial ventilation rates low is difficult because the high adrenaline state of the rescuer alters time perception, and that the rapidly refilling bag provokes a reflex in which rescuers are inclined to deliver breaths as soon as the bag inflates.

Aufferheide and colleagues (2004) reported a clinical study observing ventilation rates in cardiac arrest patients. They found that emergency medical services rescuers using a bag valve device who were trained to follow the American Heart Association (AHA) guidelines were delivering on average  $37 \pm 4$  breaths per minute, not the 10–12 breaths per minute prescribed by the guidelines. Even after the rescuers were re-trained to deliver 12 breaths per minute, they were observed delivering an average of  $22 \pm 3$  breaths per minute.

Losert and his team (2006) demonstrated excessive ventilation rates with BV devices even among trained intensivists, most of whom were basic or advanced life support instructors. Their study demonstrated that the respiration target rate was achieved only 18% of the time in patients receiving cardiopulmonary resuscitation, even when performed in a hospital setting. On average the guideline for correct ventilation rate was exceeded by 33%.

O Niell and Deakin (2007) studied BVM in comparison with a manually triggered ventilator and an automatic transport ventilator. They found that hyperventilation was common with the BVM but mainly due to high respiratory rates (ranging from 9 to 41 breaths per minute) rather than from excessive tidal volumes.

### 5.6.5.3 Excessive Inspiration Pressure

Several studies have demonstrated excessive inspiration pressures using bag valve devices which can cause barotrauma and gastric inflation.

There has been a long-standing fear that in patients with an unprotected airway excessive airway pressure caused by squeezing the bag too hard would open the oesophageal sphincter and cause inflation of the stomach, leading to potential regurgitation and aspiration into the lungs. This was a particular concern in patients being resuscitated following cardiac arrest.

A number of studies have confirmed that high pressures are often generated with a high inspiratory pressure, mask leak and gastric inflation. The latter problem has long been a major concern when ventilating through an unprotected airway such as

a pharyngeal mask. Updike and colleagues (1998) reporting a study measuring the differences between a bag valve device and a transport ventilator used with a mask found that almost 10 times the amount of air was insufflated into the simulated stomach per breath when the subjects used a bag valve device.

#### **5.6.5.4 Haemodynamic Effects of Increased Intra Thoracic Pressure When Using a Bag Valve Device**

There are a number of studies which have investigated the effects of excessive intra-thoracic pressure on the circulation when venous return to the heart is impaired. These can have serious consequences in hypovolaemic patients following physical trauma and also during CPR when high intrathoracic pressure during the decompression phase of cardiopulmonary resuscitation, decreases return of blood to the heart and impedes right ventricular function. Increased tidal volume is also known to adversely affect cardiac output.

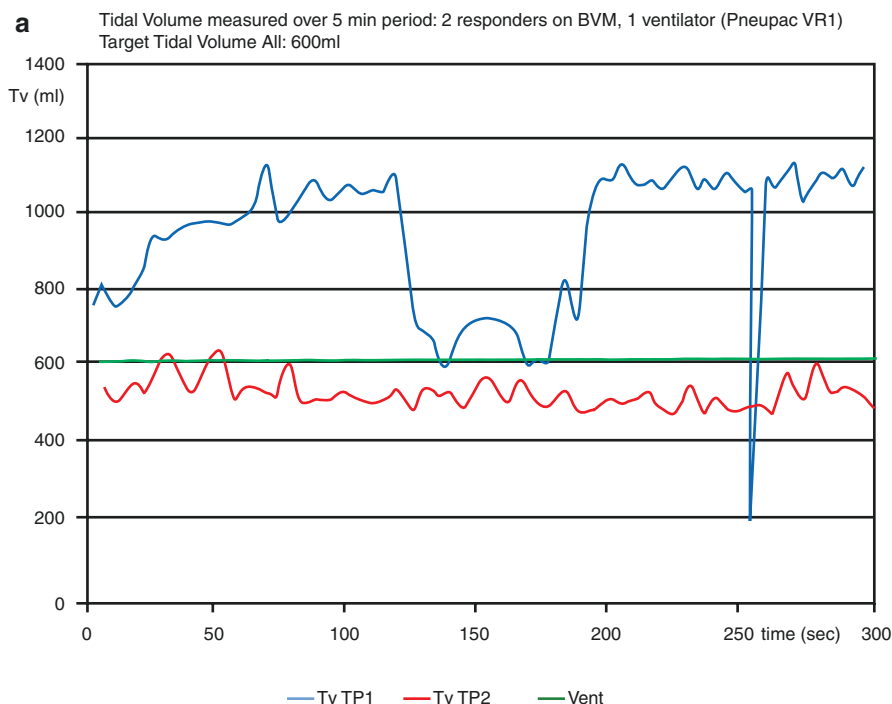
#### **5.6.6 Evaluating Ventilation Using a Bag Valve Device**

Kreft (2016, personal communication) has bench studies using a calibrated test lung to assess BV vs automatic ventilation (Fig. 5.15). Trained responders using the bag-valve device showed considerable variation in the rate and volumes of the

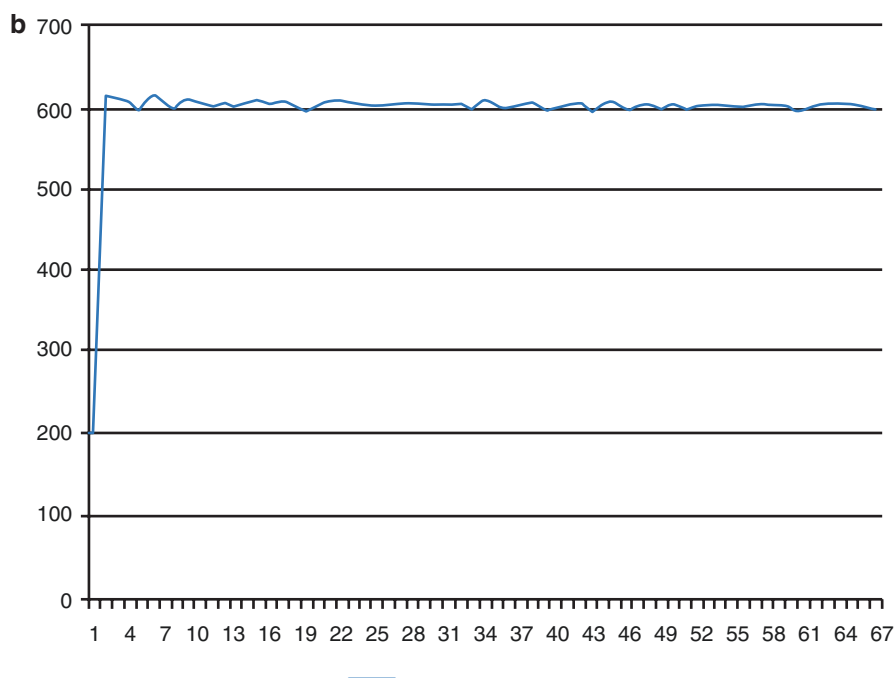
**Fig. 5.15** The Ventcheck (IngMar Medical, USA) experimental set-up. Ventilation of a mannikin using a BVM is monitored using an Ingmar test lung linked to a computer display of the ventilation parameters. (Photograph courtesy of, Smiths Medical International, Luton UK)



ventilation delivered both between individuals and in individual consistency (Fig. 5.16). This pilot study underlines the need for better training in BV management with the wider use of computer controlled test lungs and advanced manikins such as the Laerdal Sim-Man™. There is now no doubt that manual ventilation can be very variable but given that it may be the only means of ventilation available in emergency it is essential to improve training and awareness of the potential problems.



**Fig. 5.16** Ventcheck recordings of two individual BVM operators ventilating a test lung (Blue and red traces: the green trace is from an automatic ventilator used for reference) (Photograph courtesy of Smiths Medical International, Luton UK). **(a)** Variation in tidal volume delivered by BVM for two operators. There is significant variation from the target volume of 600 ml, both for individual ventilations and in the mean delivered ventilation over a period of 5 min. **(b)** Ventcheck recording with the test lung ventilated by a volume targeted automatic ventilator (Pneupac Parapac Plus) set to deliver a tidal volume of 600 ml. The individual variation in tidal volume between delivered breaths is negligible compared with the bag valve recordings of tidal volume from two subjects shown above



**Fig. 5.16** (continued)

## 5.7 Conclusions

1. A range of degrees of respiratory failure exists, from a mild form where the patient is still breathing and can be treated with free flow oxygen to complete respiratory arrest where full positive pressure ventilation is required.
2. Before effective artificial ventilation can be delivered the airway must be secured. Initially this is done positioning the head and opening the oropharynx with simple manual techniques that are part of basic life support.
3. Before artificial ventilation can begin a clear airway must be established. Airways are classified as either unprotected, where the larynx and oesophagus are still potentially in connection or protected where the trachea is isolated.
4. A range of devices exists to secure both unprotected and protected airways, such as the Guedel airway, endotracheal intubation and the laryngeal mask. The use of these with various forms of artificial ventilation depends on skill levels and experience.

5. Basic artificial ventilation in emergency can be provided using expired air ventilation. Expired air is still effective since it still contains up to 18% oxygen which is adequate to oxygenate the highly deoxygenated arterial blood following cardiac arrest.
6. Manual artificial ventilation is usually provided using a self reforming bag. These are used with a facemask (the bag–valve–mask device) in both the hospital and prehospital settings. They are widely regarded as being safe and easy to use.
7. However, the use of self–reforming bags with uncontrolled squeezing can lead to hypoventilation and hypoxia and hyperinflation which can both inflate the stomach and cause regurgitation and inhalation with an unprotected airway and also cause damaging high pressures in the lungs.
8. A number of studies have underlined the problems in the use of bag–valve devices and the need for improved training in their use.

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# Chapter 6

## Basic Principles of Mechanical Ventilation



### 6.1 Introduction

This chapter provides an introduction to the terminology and principles of positive pressure mechanical ventilation for the non-specialist. Over the past three decades artificial ventilation (AV) using basic ventilators in emergency has become widely separated from the ventilators used in the hospital setting. These have become increasingly complex and adapted by computer control to support the patient's own respiratory efforts in the best way possible. Despite the differences between ventilators used in prehospital and hospital settings the principles behind their operation are the same. The following account considers AV based upon the use of portable ventilators and the basic types of ventilation they can produce. ICU ventilators are considered in Chap. 11 but where necessary comparisons will be drawn here.

### 6.2 The Adoption of Mechanical Ventilators in Emergency Care

#### 6.2.1 Introduction

As we have seen in Chap. 3, normal breathing is a process that ventilates the lungs by creating a negative pressure inside the thoracic cavity and draws air in from the outside atmosphere. Although early forms of manual artificial ventilation (see Chap. 1) reversed this normal process by creating positive pressure to force air into the lungs using a bellows device, the earliest mechanical ventilators tried to recreate and support normal breathing by creating a negative pressure around the patient's chest. This was done by placing the patient in a chamber which created a partial vacuum and thus sucked air into the lungs by expanding the thorax. The normal

action of the intercostal muscles of the chest wall and the diaphragm was thus replaced. This type of ventilator was first developed in the 1920s and was used routinely until the 1950s. It was termed a 'cabinet ventilator' or more commonly an 'iron lung' (Chap. 1, Fig. 1.4).

A further development of negative pressure ventilation came with the creation of the cuirass ventilator, a device developed by Hauke, Eisenmenger and Bell independently. This idea has recently been reconsidered in Israel to address the logistic problem of ventilating large numbers of people at the same time following an attack using a chemical warfare agent. Mechanical positive pressure ventilation (an automated form of the basic bellows ventilation described previously came much later than negative pressure ventilation). The first device to be developed was the Draeger Pulmoflator which dates from 1911 and was used in the resuscitation of victims of mining accidents (Chap.1, Fig. 1.7). However it took until the 1950s for the next positive pressure ventilators to appear.

### ***6.2.2 Why Did Mechanical Artificial Ventilation Become So Important in the Mid Twentieth Century?***

There are three basic reasons why positive pressure artificial ventilation became so important at the middle of the last century and why it has remained so ever since

1. The world epidemic of poliomyelitis which occurred at the beginning of the 1950s overwhelmed the limited number of cabinet ventilators available. To overcome this problem the Danish anaesthetist Bjorn Ibsen used a modified anaesthetic circuit with a squeezed bag to provide IPPV (Chap. 1, Fig. 1.10) This gave rise to the concept of an intensive care unit
2. With the provision of artificial ventilation it became possible to provide intensive hospital care for seriously ill patients with trauma and overwhelming infections that caused respiratory failure. With the development of increasingly sophisticated ICU ventilators intermittent positive pressure ventilation (IPPV) became increasingly adapted to the patient's own respiratory efforts
3. The increasing use of paralysing drugs (muscle relaxants) such as curare in general anaesthesia required ventilation support. It is interesting to note that during the first use of such drugs to relax abdominal muscle and aid surgery it was not thought necessary to supplement ventilation although the muscles of the chest wall were also paralysed. The reason was that the diaphragm was much more resistant to the action of curare than the other respiratory muscles due a greater safety margin at the neuromuscular junctions controlling the transmission of signals from the brain to the muscle of the diaphragm

### 6.2.3 *Divergence Between Hospital and Emergency Ventilators*

Although early emergency ventilation (and also ventilation within the hospital) used manual methods, mechanical ventilators such as the Engstrom (Chapter 1, Fig. 1.14) were developed which used pumps driven by powerful electric motors to provide positive pressure. Such devices remained in common use in hospitals for over 20 years before being gradually replaced by smaller, more complex ventilators that were electronically—controlled and delivered compressed gas through electromagnetic valves. At the same time there was a parallel development of portable gas-powered ventilators (PGPV) which proved very effective in emergency ventilation and the transport of ventilator—dependent patients from one location to another. Box 6.1 shows the time line of the development of these devices.

#### **Box 6.1 A Time Line of Development of Portable Mechanical Ventilators**

1970s: first portable gas-powered ventilators

Initially: pressure generators

Later: volume generators

Early PGPV were non-interactive, and provided rigid ventilation suitable only for complete respiratory failure and required patient sedation and paralysis if respiratory failure was partial.

1980s: Demand valve technology

Air mix systems

1990s: Development of electrically-driven portable turbine ventilators with electronic processor control which brought many of the modes available in the ICU within the settings of prehospital and transport ventilation.

## 6.3 Artificial Ventilation of the Lungs: Terminology

The terminology of artificial ventilation has become increasingly complex with the development of modern ICU machines. However, there are basic terms and definitions which are common to both hospital and prehospital ventilation. A grasp of these is important for understanding the operation of portable mechanical ventilators. Terms used in ICU ventilation are considered in Chap. 11.

### **6.3.1 Intermittent Positive Pressure Ventilation**

Artificial ventilation of the lungs by forcing air in under positive external pressure instead of it being sucked in the normal way is termed intermittent positive pressure ventilation (IPPV).

### **6.3.2 Inspiratory and Expiratory Phases of IPPV**

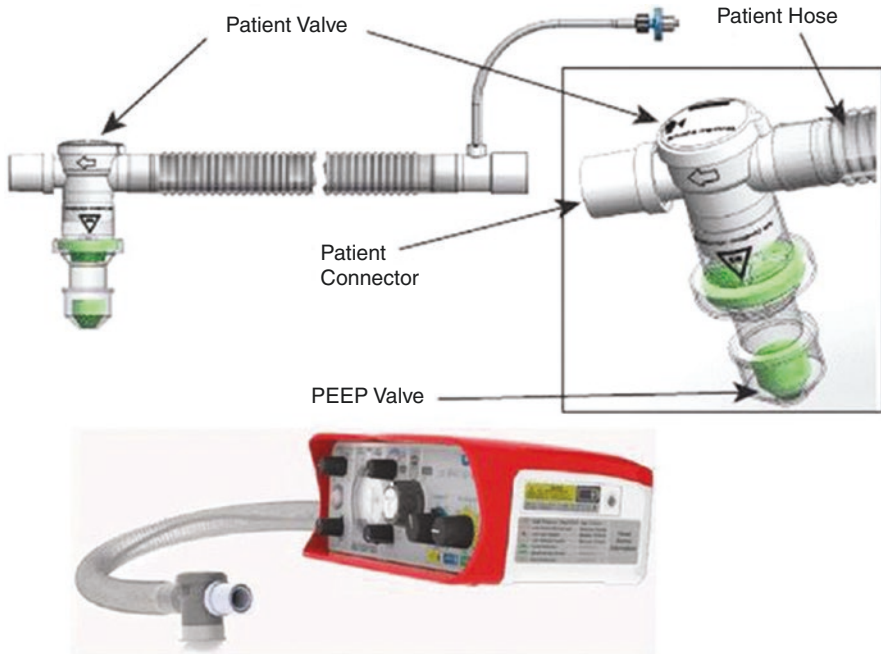
As with normal breathing, IPPV consists of two phases, inspiratory and expiratory. In artificial ventilation, the inspiratory phase (I) is active, when air under pressure is delivered to the lungs. In manual ventilation inspiration occurs when the bag is squeezed. In mechanical ventilation the way the inspiratory phase works depends of the type of ventilator used. This is considered below. At the end of inspiratory phase the ventilator stops delivering a positive pressure and the patient breathes out passively, as in normal breathing. This passive expiration (E) is ensured by the elastic recoil of the lungs and the chest wall. In some ICU ventilators there is a pause between the I and E phases, known as the respiratory pause. This is to allow distribution of gas within the lungs. Inspiratory pause is not used in non-specialist IPPV.

### **6.3.3 I:E Ratio**

Most portable ventilators have the ratio of I to E time set to 1:2, which mirrors normal breathing. Some ventilators however have the ability to alter the I:E ratio, usually with a longer expiratory time. This type of ventilation is helpful in cases of restrictive lung disease such as bronchial asthma. Most ventilators used by non-specialists have a fixed I:E ratio however. It should be noted that in most pneumatic ventilators an I:E ratio of 1:2 is only delivered over a limited range of settings of frequency and tidal volume (see below). The variation from 1:2 depends on the design and function of the ventilator. The operator should be familiar with I:E variation in the model being used.

### **6.3.4 Patient Circuit**

The connection between the ventilator and the patient is known as the patient circuit. This can take a number of forms depending on the ventilator (see Fig. 6.1) but the object of the circuit is to deliver fresh gas to the patient and evacuate expired CO<sub>2</sub>. In many pneumatic ventilators this is ensured by the provision of a patient valve which ensures the release of expired gas as close as possible to the airway



**Fig. 6.1** Patient circuit and valve for a portable gas powered ventilator (Photograph courtesy of Smiths Medical International, Luton, United Kingdom)

device being used during ventilation (usually an endotracheal tube or LMA) when the airway is secured.

### 6.3.5 Patient Circuit Filter

Many portable ventilators are fitted with a heat and moisture exchange (HME) filter to prevent the possibility of the ventilator being contaminated by the patient, if there is any infective respiratory condition. A typical filter is shown in Fig. 6.2. As the name indicates HME filters help to maintain humidification of the dry gas being delivered to the patient during IPPV.

### 6.3.6 Tidal Volume

Tidal volume ( $V_t$ ) is one of the standard lung volumes described in Chap. 2. During normal breathing at rest in an adult the  $V_t$  is about 500 ml. The  $V_t$  provided during artificial ventilation was, for many years greater than this and calculated as 10 ml/

**Fig. 6.2** An HME filter used with a portable ventilator (Photograph courtesy of Smiths Medical International, Luton, United Kingdom)



kg for adults and 15 ml/kg for infants. More recently  $V_t$  during IPPV has been reduced according to guidelines in both emergency and ICU practice. This is to prevent potential lung damage due to baro and volutrauma (see below).

### 6.3.7 *Frequency of Ventilation*

Frequency of ventilation ( $f$ ) is the number of breaths that are being provided by IPPV each minute. At rest in the adult, the normal spontaneous breathing rate is 12–20/min. In infants aged less than 2 years it is higher at 30–40/min and decreases to 20–25 between 5 and 12 years. The normal breathing rate is important in the diagnosis of respiratory failure (see Chap. 4).

### 6.3.8 *Minute Volume*

The product of  $V_t$  and  $f$  is defined as the minute volume ( $V_m$ ). In normal adults this is about 4–5 litres/min at rest. In IPPV an adequate  $V_m$  is between 8–10 litres/min. Adequacy of  $V_m$  is indicated by the level of oxygenation and the expired partial pressure of carbon dioxide ( $P_{eCO_2}$ ).  $V_m$  can be a deceptive concept. If it is calculated from a high frequency and low  $V_t$ , the  $V_m$  will be inadequate due to anatomical dead space (i.e.: most of the gas entering into the lungs does not take part in gas exchange). Also low  $f$  and high  $V_t$  values may provide an adequate  $V_m$  but may also be associated with baro and volutrauma of the lung tissue.

### 6.3.9 Dead Space

Dead space refers to the areas of the airways and lungs that do not take part in gas exchange. As discussed in Chap. 2 there are two forms (1) anatomical and (2) physiological. Anatomical deadspace is relevant to the production of type 2 respiratory failure while physiological deadspace relates to type 1 failure.

#### 6.3.9.1 Anatomical Deadspace

This comprises the gas that is in the airways and does not take part in gas exchange because it does not reach the alveoli. The average value of deadspace in the adult is 150 ml. The following factors affect anatomical deadspace.

1. It increases with size and age of the subject
2. It changes with posture of the subject, increasing in the upright and decreasing in the supine position
3. It is increased by neck extension
4. It is reduced by an artificial airway (ETT) but is increased by increasing distance from the end of the ETT to the means of evacuating  $\text{CO}_2$  in mechanical ventilation

#### 6.3.9.2 Physiological Deadspace

Physiological deadspace ( $V_d(\text{phys})$ ) results from alveoli that are ventilated but underperfused and also from areas that are over-ventilated but perfused normally. This is described as ventilation-perfusion inequality and is considered in Chap. 3.

In normal man, anatomical and physiological deadspaces are almost equal and amount to about one third of the tidal volume. The relationship between  $V_d(\text{phys})$  and  $V_t$  remains more or less constant when  $V_t$  is altered and so the physiological deadspace can be described as a fraction of the  $V_t$  (the normal ratio of  $V_d(\text{phys})$  to  $V_t$  is 0.25–0.4).

The calculation of physiological deadspace from blood gases is given by the following equation:

$$V_d(\text{phys}) = V_t (\text{PaCO}_2 - \text{PeCO}_2) / (\text{PaCO}_2 - \text{PiCO}_2)$$

where  $V_t$  is tidal volume,  $\text{PaCO}_2$  is arterial  $\text{CO}_2$  partial pressure,  $\text{PeCO}_2$  is mean expired  $\text{CO}_2$  partial pressure and  $\text{PiCO}_2$  is the inspired partial pressure of  $\text{CO}_2$ . This equation is of importance to the ventilation management of diseased lungs and is called the Bohr–Enghoff equation.

### **6.3.10 Alveolar Ventilation**

This is the volume of the  $V_t$  which is actually taking part in gas exchange and is given by  $V_a = f \times (V_t - V_d)$  where  $V_d$  is the anatomical dead space.

### **6.3.11 $FiO_2$**

The fraction of oxygen in the inspired air or gas that is being provided from a ventilator is called the  $FiO_2$ . The value in air is 0.21. In emergency ventilation with life-threatening hypoxia 100% oxygen is provided ( $FiO_2 = 1$ ). This value should be reduced to 0.4 as soon as possible to prevent possible complications from hyperoxia.

### **6.3.12 Inspiratory Pressure**

Inspiratory pressure (sometimes called patient airway pressure or PIP) is the average value of pressure of gas in the patients airway during the inspiratory phase of IPPV in pressure limited ventilation (see Sect. 6.4.3) It is usually between 15 and 20 cm  $H_2O$ .

### **6.3.13 Peak Inflation Pressure**

Peak inflation pressure refers to the value of pressure in the airway when a volume generating mode is being used (Sect. 6.4.2). Here the pressure rises until the required  $V_t$  has been delivered.

### **6.3.14 Pressure Cascade Through the Airway**

This term refers to the reduction of pressure through the airway down to the alveoli as a result of the change in flow of gas due to resistance. Pressure/volume analysis plays an important part of management of patients who are being ventilated in the ICU. In emergency ventilation however the important points are the peak airway pressure which is usually limited to 40 or 60 cm  $H_2O$  in volume generators and the mean airway pressure in pressure generators. Both these parameters are affected by the lung mechanics. Note that a set ventilation pressure does not guarantee a tidal volume.



### **6.3.15 *Hyperventilation***

Hyperventilation occurs when too high a minute volume is delivered to the patient. This can arise as a result of tidal volume being too high, ventilation frequency too high or both. If the hyperventilation is delivered at a normal inspiratory pressure the result will be to lower the expired CO<sub>2</sub> level causing a respiratory alkalosis. This can cause problems with muscle action. If the hyperventilation is associated with too high an inflation pressure the following can occur:

#### **6.3.15.1 Barotrauma**

Barotrauma occurs when the air sacs of the lung burst due to the high inflation pressure causing a pneumothorax (the presence of air between the pleural membranes lining the thorax). This is a serious emergency and must be treated immediately with chest drainage.

#### **6.3.15.2 Volutrauma**

This condition was identified in ICU some years ago and led to a major revision of the way patients are ventilated, using small tidal volumes which are accompanied by an increase in the CO<sub>2</sub> level. This type of ventilation (known as permissive hypercapnia) is not used in emergency ventilation where there is a risk of life-threatening hypoxia but has a role in the transport of patients with serious lung conditions such as acute respiratory distress syndrome (Chap. 11).

Resuscitation guidelines have been altered to provide smaller V<sub>t</sub> during emergency and it is likely that the prevention of volutrauma during emergency ventilation will gain increasing importance in coming years. The important point is to avoid high V<sub>t</sub> and PIP and titrate the ventilation according to oxygen and carbon dioxide levels, providing the ventilation that is required for the clinical situation. Barotrauma and volutrauma can be prevented by ensuring that the PIP is not too high. This is ensured on portable ventilators by a relief valve built into the device which releases the excess pressure at 40 cm H<sub>2</sub>O (60 cm is adopted in the USA). Some ventilators allow the user to control the blow-off pressure.

### **6.3.16 *Hypoventilation***

Hypoventilation occurs when there is a failure of alveolar ventilation due to too small a V<sub>t</sub> being delivered to the patient. In the worst case the V<sub>t</sub> falls below the anatomical dead space and there is no ventilation at all. This can occur due to inappropriate settings of the ventilator or following disconnection of the ventilator from

the patient through the patient circuit. It can also occur as a result of serious leaks around the ETT. Hypoventilation is rectified by careful clinical observation of the ventilated patient, backed up by hypoventilation alarms which are incorporated into many emergency and transport ventilators.

### ***6.3.17 Positive End-Expiratory Pressure***

Positive end-expiratory pressure is produced by maintaining a positive pressure in the patient circuit at the end of inspiration. This can be set by the operator and is common to both portable and ICU ventilators. PEEP has an important role to play in improving oxygenation by opening up the lung and increasing the functional residual capacity (see Chap. 3). It is discussed in more detail in Sect. 6.5.3 below.

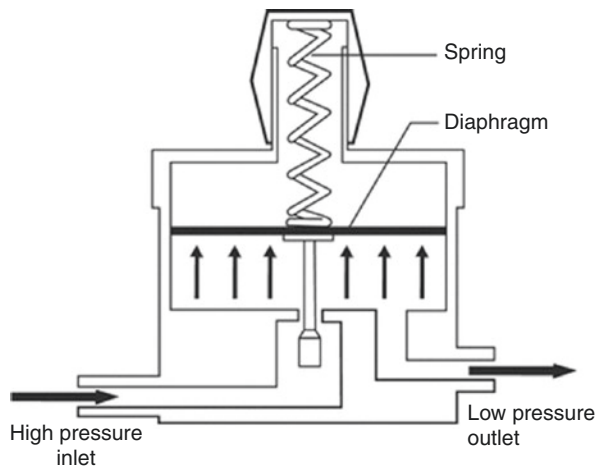
### ***6.3.18 Air Mix***

Portable pneumatic ventilators are driven by compressed oxygen and originally could only deliver 100% oxygen to the patient. This concentration is suitable for emergency resuscitation where severe hypoxia is present but is contraindicated for longer term ventilation and where a patient has returned to spontaneous circulation following a cardiac arrest. The invention of the air mix system allowed a reduced concentration of 40–50% oxygen to be delivered to the patient by entraining air into a flow of oxygen through a venturi system (Chap. 7) and mixing it with the gas being delivered to the patient. On portable ventilators the air-mix valve can be set to either 100% or to 40–50% depending on the model.

### ***6.3.19 Reducing Valve***

The pressure in a compressed oxygen cylinder is about 2000 psi (3000 psi if the cylinder is made of lightweight Kevlar carbon fibre material). This pressure is too high to drive a portable gas-powered ventilator and so must be reduced to about 40–60 psi before being fed to the ventilator through a high pressure supply hose. The high pressure gas from the cylinder is fed to a device called a reducing valve shown in Fig. 6.3. This contains a diaphragm which is activated by the high pressure gas and creates a lower pressure on the other side by means of a connecting rod which opens and closes the flow of gas from the cylinder. The regulating valve is set by the operator who sets the pressure applied to the diaphragm via a strong spring. The reduced pressure is then fed to the ventilator which contains a regulator system to control the feed pressure to exactly the pressure required for the ventilator to function.

**Fig. 6.3** A basic reducing valve (reproduced with permission from Aitkenhead AR and Smith G (eds) *Textbook of Anaesthesia* (second edition), Churchill Livingstone (Elsevier), London, 1990)



The high pressure from the cylinder enters the valve and forces the flexible diaphragm upwards. The diaphragm is connected to an entry valve which is forced upwards and closes off the high pressure flow. The force required to do this is controlled by the spring on the other side of the diaphragm. This spring can be tightened manually. The more it is tightened the greater will be the force required. The relationship between the low ( $p$ ) and high ( $P$ ) pressures is approximately related to the areas of the diaphragm ( $A$ ) and the valve seating ( $a$ ) by the relationship:  $p/P = a/A$ . When the spring is tightened a force  $F$  is produced that offsets the closing of the entry valve. Therefore the reduced pressure may be controlled by increasing the tension in the spring.

### 6.3.20 Manometer

Many portable ventilators contain a pressure gauge or manometer which shows the pressure of the gas being fed to the patient during the inspiratory phase. The gauge is usually calibrated in  $\text{cm H}_2\text{O}$  which is a very low unit of pressure. The manometer allows the ventilator operator to see if the pressure being delivered to the patient is too high (this often means a kinked ETT or patient circuit) or too low, where the most likely explanation is a disconnection of the patient circuit from the ventilator.

### 6.3.21 CMV/Demand

This control switches the ventilator between controlled mandatory ventilation with an override if the patient's own breathing efforts are adequate or demand where IPPV is switched off and the patient receives gas from the ventilator only when he

**Table 6.1** Oxygen cylinder capacities

Cylinder size	C	D	E	F	G	H
Capacity (litres at atmospheric pressure)	170	340	680	1360	3400	6800

takes a breath. The advantage of demand breathing is that oxygen is only delivered to the patient during the inspiratory phase of breathing and not during both inspiratory and expiratory phases as in the case of oxygen being delivered by constant flow from a face mask.

### 6.3.22 *Oxygen Cylinders*

Oxygen cylinders contain gas at a high pressure (2200 psi or 3000 psi for cylinders made of Kevlar carbon fibre). The high pressure is reduced to one to two atmospheres (14–28 psi) to provide free flow oxygen via a regulator valve or to drive a PGPV. Cylinders are available in several different sizes. The contents of the cylinder refer to the volume of the compressed gas would occupy at atmospheric pressure. Table 6.1 gives details of a range of oxygen cylinder sizes. The most commonly used sizes in portable ventilation are D and E.

## 6.4 How Mechanical Ventilators Work

Although ventilators have become increasingly complex over the past 50 years the underlying principle of their operation remain the same, that is to pump air into the lungs and allow it to escape passively. As noted above, this is the reverse of normal physiological breathing which depends on creating negative pressure inside the thorax. Early mechanical ventilators, designed for use in hospital relied either on an electric motor driving a pump or a piston device (example: the Engstrom ventilator) or on compressed gas from an anaesthetic machine (for example the Manley ventilator).

Today, ventilators can broadly be classified as (1) simple devices designed to operate in the prehospital or emergency setting, which can be operated by non-specialists and (2) more complex ventilators whose function is to provide respiratory support in the hospital settings of the operating theatre and intensive care unit. However, this classification is increasingly complicated by the development of complex battery-driven, electronic ventilators which can operate in the prehospital setting but which have most of the ventilation functions found in ICU devices.

IPPV provided by mechanical ventilators can take several forms or modes which will be discussed later. Since respiratory failure can be either complete or partial,

where the patient still has some breathing effort, IPPV may also be total or partial depending on how much respiratory effort the patient is able to provide. Total ventilation support is required where the patient is in complete respiratory arrest (see Chap. 4) or where the patient has been deliberately paralysed to aid surgery, as in general anaesthesia. In cases where the patient is still partially breathing, ventilation support must adapt to the patient's own efforts and support them. This is usually the case in the ICU where complex, computer controlled devices adapt closely to the patient's breathing efforts and support them (see Chap. 11).

In IPPV for emergency respiratory arrest, the lung mechanics (compliance and resistance) may be essentially normal if there is no pre-existing respiratory disease. This is the case for example in head injury accompanied by respiratory arrest. In ICU ventilation, lung mechanics are usually abnormal with decreased compliance and increased resistance. This is the case in the acute respiratory distress syndrome (ARDS (see Chap. 11)).

The two settings where IPPV is used, prehospital and hospital require different approaches to artificial ventilation which are discussed below. Overall, prehospital ventilation is done using ventilators which are simple to operate and which provide essential and reliable ventilation support for patients in danger of hypoxia following respiratory arrest. Respiratory support is usually complete but some adaptation to the patients own respiratory efforts is possible. In the hospital the ICU ventilators are now able to provide a wide spectrum of ventilation support ranging from complete IPPV through to applied pressure support for a patient who is still breathing but with reduced effectiveness.

### ***6.4.1 The Respiratory Cycle: The Essential Function of a Mechanical Ventilator***

Mapleson in 1969 described four phases of the function of a mechanical ventilators that are still applicable to both modern simple and complex devices. This is known as the respiratory cycle which consists of:

1. The inspiratory phase
2. Cycling between the inspiratory (I) and expiratory (E) phase
3. The expiratory phase
4. Cycling between the E and I phase

During the inspiratory phase gas under positive pressure is delivered to the lungs according to whether a preset volume is required (volume generator) or whether a preset pressure within the patient's airway has been achieved (pressure generator). An understanding of the difference between these two overall forms of ventilation is important for the selection and use of portable mechanical ventilators.

### **6.4.2 Volume Generators**

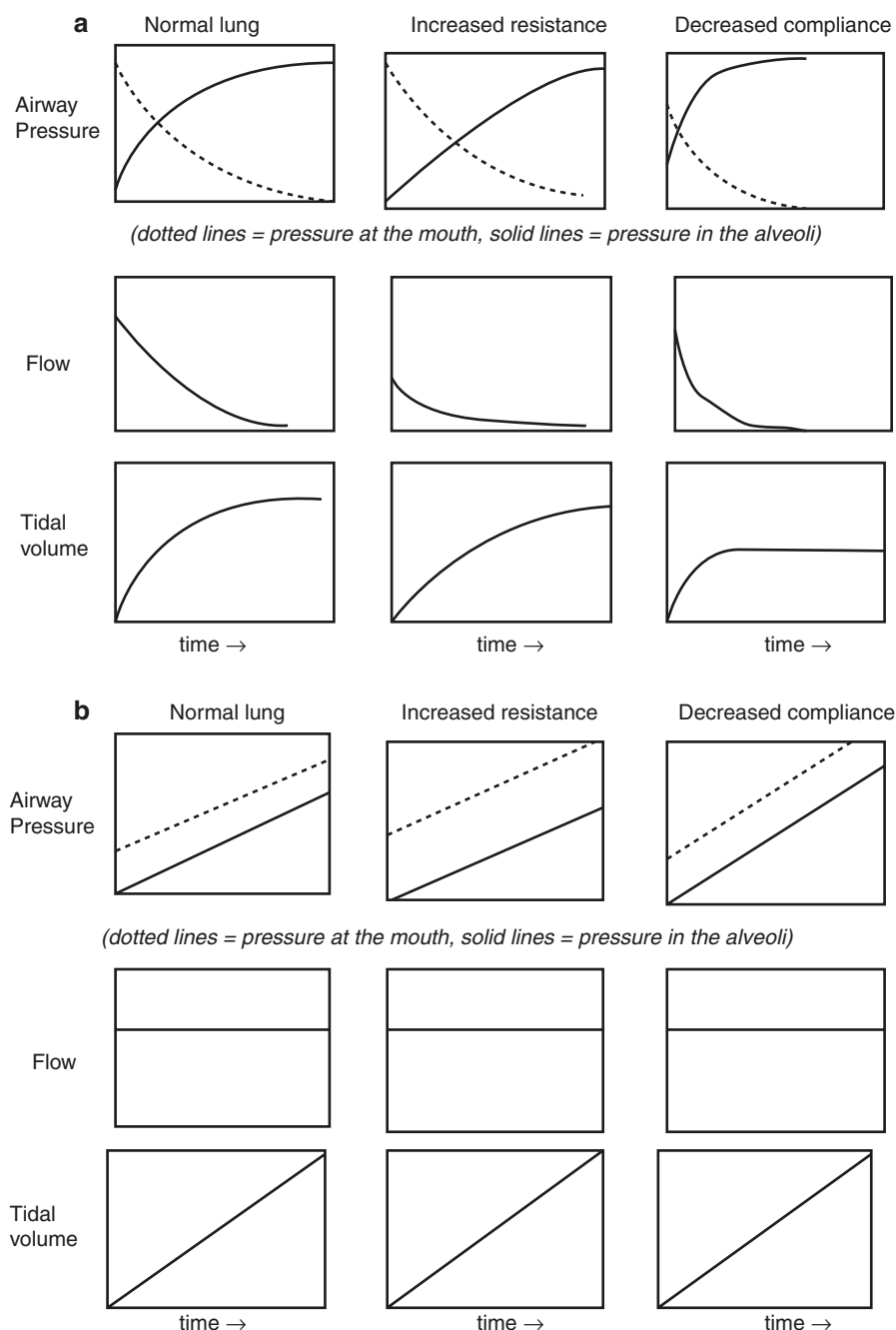
In these ventilators the desired tidal volume to be delivered to the patient is set by the operator and is a function of the flow rate of the gas within the circuit. This volume is generated during the inspiratory phase by a set flow of gas and the airway pressure rises. The volume delivered is independent of compliance and resistance changes within the lungs and the delivered pressure rises accordingly to ensure that the preset  $V_t$  is delivered. The original volume (or flow) generators were pump devices where a motor compressed a bellows. More modern devices ensure  $V_t$  through gas flow by the provision of compressed gas which is controlled either pneumatically (as in many portable ventilators) or by computer boards controlling electromagnetic valves, as in the case of ICU ventilators. The latter are capable of many more ventilation modes than the former. However, many of these are not appropriate for emergency ventilation.

### **6.4.3 Pressure Generators**

These are ventilators which provide a constant pressure during the inspiratory phase. The volume delivered is not constant and depends on the mechanical characteristics of the lungs. Pressure generators were also originally mechanical and consisted essentially of a weight acting on a concertina bag. As with volume generators modern pressure generators are now pneumatically or computer controlled.

Pressure generation during the I phase is a feature of much of the ventilation in the ICU where it is used to support the patient's own breathing efforts. In this setting it has the advantage of being more accepted by the patient and helps in discontinuing IPPV when indicated. The problem of pressure generation is that it cannot guarantee an adequate  $V_t$  in situations where the lung mechanics are abnormal or where a guaranteed  $V_t$  is required due to life-threatening hypoxia. They are therefore not suitable for most emergency ventilation, with the notable exception of neonatal and infant ventilation where flow generators are contraindicated due to the fragility of the lung tissue.

In both volume and pressure generators there are safeguards against the production of excessive pressure within the lungs (barotrauma). In volume generators this is achieved by having a safety valve which activates at a preset pressure (usually 40 cm  $H_2O$ ). In pressure generators the maximum pressure that can be achieved is set by the operator. The characteristic pressure, flow and tidal volume waveforms for normal and diseased lungs produced by volume and pressure generating ventilators are shown in Fig. 6.4.



**Fig. 6.4** Lung filling with a constant pressure ventilator (**a**) and a volume preset ventilator (flow generator). (**b**) Each of the set of two figures shows the flow the delivered volume and pressure generated in the airway over the inspiratory period. Curves are shown for normal lung mechanics and for increased airway resistance (e.g. chronic obstructive airway disease) and for decreased compliance (e.g. infections and ARDS)

### **6.4.4 *Cycling Between the I and E Phase***

There are three ways in which ventilators can change from inspiration to expiration at the end of the I phase. These are:

#### **6.4.4.1 Time Cycling**

In time cycling the change from I to E occurs after a fixed time and is not influenced by the state of the patient's lungs. This is the most common method of cycling used in modern ventilators, both portable and hospital devices. In hospital ventilators the I phase is often followed by a short pause before expiration (the inspiratory pause) which improves gas distribution within the lungs. Time cycling is associated with preset volume in the case of many portable gas-powered ventilators.

#### **6.4.4.2 Volume Cycling**

This type of cycling was used in older ventilators. Here the ventilator cycles between I and E when a predetermined  $V_t$  has been delivered. The duration of the I phase is determined by the flow rate of gas during the I phase. In older machines it was activated simply by a cam which stopped the bellows before the end of its travel.

#### **6.4.4.3 Pressure Cycling**

In pressure cycling, the change from the I to E phases occurs when a preset pressure is reached. The time taken to reach this pressure is determined by the lung mechanics. Therefore the time taken to reach a preset pressure and therefore the duration of the I phase is much shorter when the airway resistance is high.

#### **6.4.4.4 Expiratory Phase**

The E phase of the respiratory cycle is essentially passive, as it is in normal breathing. In the past some ventilators tried to improve performance by providing a negative pressure to suck gas out of the lungs during the E phase but this practice has been discontinued in modern ventilators. During the E phase pressure in the patient's airway ( $P_{aw}$ ) falls almost to atmospheric. However, both portable and hospital ventilators are able to alter this pattern by maintaining a small pressure (usually between



5 and 15 cm H<sub>2</sub>O) at the end of expiration. This is known as positive end–expiratory pressure (PEEP). PEEP is widely used in both prehospital, transport and hospital ventilation to improve the oxygenation of the blood by V/Q improvement. Sometimes, PEEP will be generated as a result of the lack of time during expiration for the lungs to empty. This is termed autoPEEP. PEEP is discussed further in Sect. 6.5.3.

#### **6.4.4.5 Cycling from the Expiratory to the Inspiratory Phase**

This is achieved by time cycling ie the change from E to I occurs after a set time. This can be varied on most ICU ventilators and on some pneumatic ventilators (e.g. the Pneupac VentiPac). This provides a variable I: E ratio which is useful in the management of conditions where expiration is affected by lung conditions eg asthma. However, in most portable ventilators the I:E ratio is fixed at its normal physiological value of 1:2.

#### **6.4.5 Triggering: Patient Activation of the Inspiratory Phase**

As noted above, in modern hospital ventilators there is an emphasis on adapting the patient's own breathing efforts to the function of the ventilator. This is done by providing the ventilator with a system that detects the patients own breath and activating a ventilator breath to support the spontaneous efforts. This is termed 'triggering' and is done in the following ways:

1. by detecting a change in the patient airway pressure to sub-atmospheric
2. by detecting a flow of gas into the lungs
3. by detecting a volume change as a result of the patient's efforts to inhale

These three methods of triggering are supplemented by time–triggering where supportive breaths are delivered to the patient at regular preset intervals, independent of the patients own efforts. This is used in simple synchronised adaptive modes found on portable ventilators such as SIMV and SMMV which are discussed below.

##### **6.4.5.1 Why Is Triggering Important?**

Triggering allows interactivity between the ventilator and the patient's own breathing efforts. Early ventilators, including portable devices could not synchronise with

the patient if there was partial respiratory failure. IPPV was only possible in total respiratory failure or if the patient was paralysed or heavily sedated with opioids which suppress the action of the respiratory centre in the medulla of the brain. Modern hospital ventilators, which have electronic sensors and computer control use very sensitive and flexible triggering usually based upon flow triggering. This allows complete interactivity with the patient's own respiratory efforts. In portable ventilators patient triggering was not possible until the 1980s when demand valves with pressure triggering were introduced by the Pneupac company in the UK. The demand valve, which can detect small negative changes in patient airway pressure allows synchronisation with the patients breathing efforts or suppression of the action of the ventilator completely if the patient's breathing is at a normal level. Thereafter, the patient is supplied with oxygen through the demand valve.

## 6.5 Modes of Positive Pressure Ventilation

6.5 A ventilation 'mode' describes the characteristics of the ventilation provided by a ventilator and also how it interacts with the patient's own respiratory efforts. Ventilation modes can be divided into (1) controlled and (2) assisted ventilation in terms of the work of breathing done by the ventilator and the patient. The terminology describing ventilation modes is often complicated and non-standardised. Although there are a limited number of true modes of ventilation a confused terminology has arisen, often driven by manufacturers for marketing purposes, to describe variations (often very slight) from the basic modes. Here we consider only the basic modes of ventilation found in portable ventilators for emergency and transport use. These range between total ventilatory support and spontaneous breathing. In more complex electronically controlled modern ICU ventilators ventilation is usually based upon support for the patient's own respiratory efforts using sophisticated sensors and generated patterns of ventilation (see Chap. 11).

### 6.5.1 *Ventilation Modes Classified According to the Work of Breathing*

As we have seen earlier, intermittent positive pressure ventilation is necessary according to how much respiratory support is required. The diagnosis of respiratory failure was the subject of the previous chapter. To decide whether a patient requires ventilation following injury or during illness, the following points must be considered:

## 1. Are there signs of respiratory failure?

- shallow or slow breathing
- abnormal breathing forms
- reduced oxygen saturation in the blood
- increased end-tidal and arterial CO<sub>2</sub>

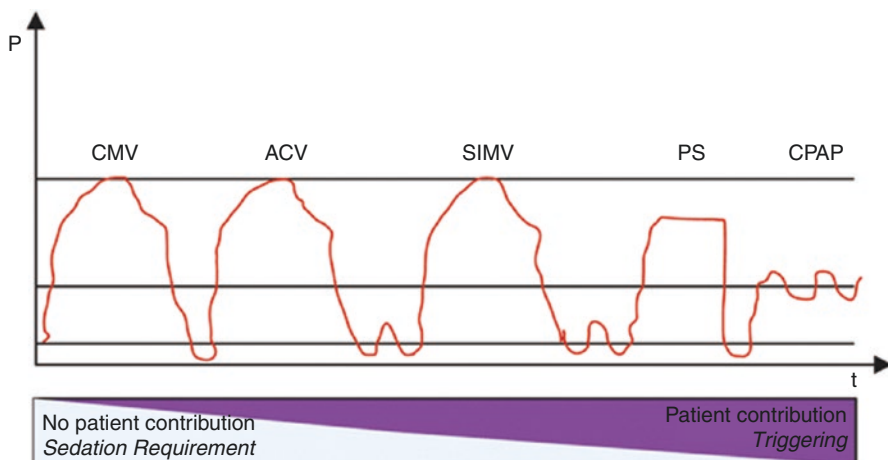
## 2. Is the patient able to initiate a breath?

## 3. Is the patient able to perform the required work of breathing?

Answers to these questions determine whether the patient will require complete or partial respiratory support from the ventilator. In emergency situations early artificial ventilation is often required quickly based upon a rapid assessment of the respiratory status. In this situation portable ventilators are used which can provide complete ventilation but can then detect and adapt to any breathing effort the patient may make.

### 6.5.2 The Spectrum of Modes of Ventilation

Ventilation modes can best be understood as a spectrum describing the work of breathing done by the ventilator and by the patient. This is shown diagrammatically in Fig. 6.5.



**Fig. 6.5** The spectrum of basic modes of artificial ventilation (Authors image). *CMV* Controlled Mandatory Ventilation, *ACV* Assist Control Ventilation, *SIMV* Synchronised Intermittent Mandatory Ventilation, *PSV* Pressure Support Ventilation, *CPAP* Continuous Positive Airway Pressure

### **6.5.2.1 Controlled Mandatory Ventilation (CMV)**

In this mode, ventilation is provided entirely by the ventilator. The patient is in complete respiratory failure, cannot initiate inspiration and contributes nothing to the work of breathing. This is the situation at the left hand end of the spectrum shown in Fig. 6.5.

### **6.5.2.2 Assisted Controlled Ventilation (ACV)**

In this mode, ventilation is still essentially automatic but the patient plays a greater part in the work of breathing.

### **6.5.2.3 Synchronised Mandatory Ventilation (Mixed Mode Ventilation)**

In this mode the patient's respiratory efforts are synchronised with ventilations provided automatically by the ventilator. Subdivisions of this mode of ventilation exist to describe (1) situations where the patient's own ventilations are supplemented by ventilations from the ventilator to provide an assured minute volume to provide adequate oxygenation of the blood. (2) where the patient's own respiratory efforts determine the start of the inspiratory phase of the ventilations delivered by the ventilator. This type of ventilatory support is called assist control ventilation and is most commonly found in hospital ICU ventilators.

In ventilation using portable ventilators there are three standard mixed modes of ventilation. These are:

1. Intermittent mandatory ventilation (IMV)
2. Synchronised intermittent mandatory ventilation (SIMV)
3. Synchronised mandatory minute volume (SMMV)

#### **Intermittent Mandatory Ventilation**

Intermittent mandatory ventilation was an early mixed mode of ventilation, originally conceived for weaning patients off a ventilator in the hospital setting, after a prolonged period of artificial ventilation. In IMV, the patient breathes spontaneously but has an inadequate minute volume (MV). The MV insufficiency is compensated by the ventilator which administers breaths of predetermined volume and duration. The disadvantages of IMV are (1) the set rate of automatic ventilation must be below the patient's spontaneous breathing rate (2) since the automatic ventilation rate is fixed the patient may have to work against the ventilator if spontaneous ventilation expiration takes place at the moment when the ventilator is commencing an automatic inspiration.

### Synchronised Intermittent Mandatory Ventilation (SIMV)

Synchronised intermittent mandatory ventilation (SIMV) was introduced during the 1980s to overcome the problems of IMV. It is found on a number of modern portable ventilators. In SIMV, any breath taken by the patient is synchronised with a breath provided by the ventilator. The ventilator breath is initiated by a trigger (which is usually a pressure drop detector) which detects the patient's own respiratory efforts. In this situation, the ventilator breath may be triggered earlier than would be expected in simple IMV. The result is therefore an increase in ventilation frequency. In ICU practice SIMV has been overtaken by assisted ventilation modes which rely on sophisticated electronic sensors and processors. The value of SIMV in emergency and transport ventilation is that it allows ventilation of a patient who is in partial respiratory failure and who might use the remaining respiratory effort to work against mandatory ventilation (a situation known as 'fighting the ventilator.')

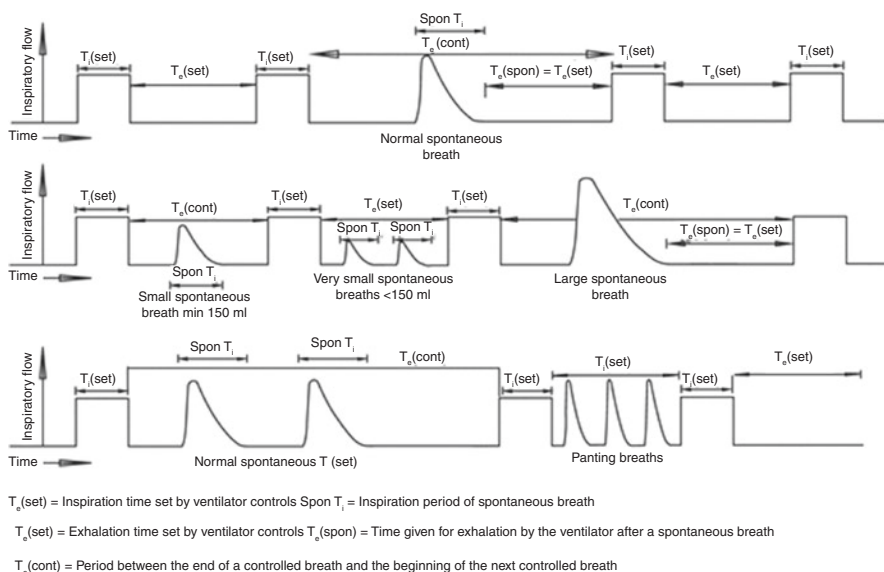
### Synchronised Mandatory Minute Volume (SMMV)

IMV and SIMV work on the principle that the respiratory minute volume needed to provide adequate ventilation is divided between the ventilator and the patient. Thus, if the patient does not breathe adequately or stops breathing altogether he receives compensatory ventilation from the ventilator to provide an adequate minute volume. Synchronised mandatory minute volume mode (SMMV) works on a different principle from SIMV in that it is the overall minute volume and not time that is the determining factor in deciding the need for supplementary ventilation from the ventilator in a patient with partial respiratory failure. In classical SMMV the ventilator measures the volume of gas being breathed by the patient over a set period of time (e.g. 10s) and compares this with a set minute volume. If there is a discrepancy between the actual minute volume and the set delivered minute volume, an automatic ventilation is delivered which corrects the difference, thus ensuring an adequate minute volume.

### The Pneupac SMMV Mode

One major problem with conventional MMV is that the ventilator cannot distinguish between dead space ventilation (i.e. where the tidal volume is less than about 150 ml in the adult but with a frequency sufficient to achieve a correct minute volume) and alveolar ventilation (where there is higher tidal volume, sufficient to move gas in and out of the alveoli, and a lower ventilation frequency). To overcome this problem, the Pneupac company have produced their modified version of version of SMMV which works as follows:

When the ventilation is set to provide CMV/SMMV, CMV continues according to the settings of tidal volume and ventilation frequency unless the patient takes a breath which the ventilator detects as being with a flow rate of at least 15



**Fig. 6.6** The Pneuapac SMMV mode. Automatic ventilation (square waves) is suppressed if the patient takes a sufficient breath by extending the expiratory time. Small and panting breaths are ignored

litres/min (on a Pneuapac Parapac Plus ventilator this is equivalent to a tidal volume of at least 450 ml). If such a breath is taken, the expiratory time of the CMV is prolonged for a further period of the set  $T_e$ , unless the patient takes another adequate breath when the  $T_e$  prolongation is repeated. If there is no further adequate spontaneous breath or if the tidal volume of the breath is between 150 and 40 ml, or if there are small rapid panting breaths of less than 150 ml the ventilator automatically reverts to the set CMV mode designed to deliver an adequate minute volume ( $V_t \times \text{frequency}$ ). This form of SMMV is shown diagrammatically in Fig. 6.6.

### 6.5.2.4 Assisted Spontaneous Breathing (ASB)

In this mode the patient is breathing spontaneously but with insufficient rate or depth to ensure a sufficient tidal volume. The work of breathing is also too much for the patient who tires, making the situation worse. The ventilator therefore works only by supporting the patient's own respiratory efforts. The mode is most usually known as assisted spontaneous breathing (ASB) or pressure support ventilation (PSV). It should be noted that these modes are usually used in the ICU setting and not in emergency ventilation.

### 6.5.2.5 Spontaneous Breathing Support

Spontaneous breathing support is not specifically a positive pressure mode since all the work of breathing is done by the patient. However, to improve oxygenation in lungs that may not be exchanging oxygen with the blood adequately (for example where there are shunts or fluid within the alveoli) spontaneous breathing can be made more effective by arranging a constant positive pressure against which the patient breathes during inspiration and expiration - continuous positive airway pressure (CPAP) or with different pressures during inspiration and expiration (BiPAP). It should be noted that CPAP and BiPAP do not provide intermittent positive pressure ventilation and cannot be used unless the patient is breathing.

### 6.5.3 Positive End-Expiratory Ventilation (PEEP)

The effectiveness of AV can be improved by applying a positive pressure to the patient during the expiratory phase. This is known as positive end expiratory pressure (PEEP) and is widely used both in hospital and prehospital practice. In this mode the pressure during expiration in CMV is not zero but expiration takes place against a set pressure, usually between 0 and 15 cm H<sub>2</sub>O. The purpose of PEEP is to increase the functional residual capacity of the lungs and thus the efficiency of oxygenation of the blood. PEEP is used in emergency ventilation with portable ventilators and also in the ICU. The increase in the FRC with PEEP makes the mixing of fresh gas and expired gas in the lungs more efficient and avoids wide swings in oxygenation which would otherwise occur.

During intermittent positive pressure ventilation the inspiratory phase is active, with the lungs being inflated by the application of a positive pressure through the patient's airway while the expiratory phase is passive, with air being exhaled from the lungs through the passive recoil of the chest wall and the elastic lungs. During expiration the pressure against which the gas moves out of the lungs is almost that of the atmospheric pressure surrounding the exterior of the patient and which is in direct contact with the airway. In PEEP however, the pressure against which the expiratory gas flows out of the lungs is slightly greater than that of the atmosphere (being between 0 and 15 cm H<sub>2</sub>O). In the patient circuit shown in Fig. 6.7 this is

**Fig. 6.7** The Pneupac Spring-loaded PEEP valve (Photograph courtesy of Smiths Medical International, Luton, United Kingdom)



achieved by placing a simple spring loaded valve in the expiratory section of the patient valve so that expiration has to take place against the force set by the spring. In more complex electronic ventilators PEEP is generated by introducing a flow of gas into the expiratory arm of the ventilator during expiration.

### 6.5.4 Continuous Positive Airway Pressure (CPAP)

At the right hand end of the ventilation spectrum shown in Fig. 6.4 the patient is providing all the work of ventilation through normal breathing and the ventilator is not adding anything. However, this normal ventilation can be again made more efficient as with PEEP by applying a positive pressure against which the patient has to breathe. This is known as continuous positive airway pressure (CPAP). CPAP, as its name implies differs from PEEP in that the positive pressure is provided to the patient's airway during both inspiration and expiration. This achieves the following.

1. It makes the work of inspiration easier
2. It makes the work of expiration harder but helps to open up (recruit) alveoli in the lungs that may be non-ventilated (i.e. shunted). This improves overall oxygenation. CPAP can also help to open up collapsed bronchi and improve ventilation to the parts of the lung they serve.
3. In situations where there is type 1 respiratory failure such as congestive heart failure where there is a build-up of fluid in the alveoli CPAP helps to push the fluid back into the circulation via lymphatic drainage of the lung interstitial space according to the Starling equation. This improves the diffusion of oxygen into the pulmonary capillaries. CPAP may be used in this way in both the emergency prehospital and hospital settings.

CPAP is usually provided using a special circuit driven by a flow of compressed oxygen as shown in Fig. 6.8. This is delivered to the patient using a tight fitting face mask. Breathing with CPAP has been compared to the sensation of breathing against a very strong wind. CPAP is not suitable for all patients and tolerance may be

**Fig. 6.8** An adult CPAP circuit (Photograph courtesy of Smiths Medical International, Luton, United Kingdom)





variable. CPAP is not a ventilation mode and if the patient stops breathing it does not provide AV. It is essential that any patient being given CPAP must be fully conscious and compliant and able to tolerate the tight fitting mask required.

### **6.5.5 Bilevel Positive Airway Pressure (BiPAP)**

Bilevel Positive Airway Pressure (BiPAP) is a form of CPAP used in the ICU where the applied pressures during the inspiratory and expiratory phases are different, leading to better patient tolerance. BiPAP is discussed further in Chap. 11.

## **6.6 Conclusions**

1. In this chapter we have considered a basic classification of how all mechanical ventilators provide intermittent positive pressure ventilation.
2. This is based on the respiratory cycle which is common to all ventilators whether simple or complex.
3. The type of IPPV produced by a ventilator is termed a 'mode.' Many different modes exist and are sometimes given trademarked names by manufacturers. However a basic classification exists which has been used here. The essential function of different modes is to adapt to the degree of ventilatory effort a patient in respiratory failure may still have. In emergency ventilation complete respiratory support is required for a patient who is not breathing.
4. A spectrum of modes exist between complete ventilator support and patient who is still breathing spontaneously but with reduced efforts.
5. In the hospital ventilatory support is usually provided using pressure support found on ICU ventilators but an increasing number of transport ventilators now also have this function.
6. PEEP can improve the ventilation efficiency in patients who are still being ventilated. The equivalent support in a spontaneously breathing patient is CPAP but it must be noted that this is not a ventilation mode. Before receiving CPAP a patient must be seen to be breathing adequately.
7. The many complex modes found on modern hospital ventilators make their operation the realm of specialist therapists and bioengineers. These are considered further in Chap. 11 and Appendix E. In the next chapter we consider the operation and use of portable ventilators which can be used by non-specialists.

## **Suggestions for Further Reading**

- Hasan A. Understanding mechanical ventilation: a practical handbook. 2nd ed. London: Springer; 2010.
- Papadakos PJ, Lachmann B. Mechanical ventilation: clinical applications and pathophysiology. Philadelphia, PA: Saunders (Elsevier); 2008.

# Chapter 7

## Portable Mechanical Ventilators



### 7.1 Introduction

Portable ventilators have been increasingly used in both hospital and prehospital practice for nearly 40 years. They may be defined as mechanical ventilators which are easily-carried and can be used in a wide range of non-hospital settings, away from services such as mains power and piped gases. Originally these ventilators were powered by compressed gas, and followed from the original concept of the Drager Pulmoflator, which dates from 1909. The history of portable gas-powered ventilators (PGPV) was outlined in Chap. 1. Today a wide range of portable ventilators exists, ranging from small hand held devices designed for emergency resuscitation through to complex electronically controlled machines that are effectively miniaturised versions of ventilators used in the ICU. While many portable ventilators are still powered by compressed gas there has been an increasing shift towards devices that depend on both compressed gas and battery power, because of an increasing customer demand for graphic displays.

Portable ventilators are used in emergency management of respiratory failure either replacing bag valve ventilation or continuing from it when the initial emergency has been stabilised. They are used also in the transport of ventilator-dependent patients from one location to another. This ranges from a few metres inside the hospital to many thousands of kilometres during medical evacuation flights. Transport ventilation is a specialised use of portable ventilators and is considered in the next chapter. Note that in the USA portable ventilators are sometimes known as automatic transport ventilators (ATV) whether or not they are being used in a transport role.

Chapter 5 considered the provision of emergency artificial ventilation at a basic level, either as rescue breathing or manually using a bag-valve device. This chapter concerns the function and classification of portable ventilators which provide the next stage in emergency ventilation. It also covers the checks required and the

protocols to be followed. It is written for the non-specialist who may not be very familiar with mechanical ventilation or who may use portable ventilators only occasionally. It is important to note that the clinical guidance given can in no way replace practical instruction and hands-on experience and the intention is to provide advice that will supplement these essential points. A basic understanding of how portable ventilators work and the checks that should be carried out is essential to the safe use of these devices. These are covered with reference to a small number of standard portable ventilators that are on the market. A comparison of the technical characteristics of such ventilators is presented in appendices 3 and 4. How to choose a ventilator from the bewildering range that is currently available on the market is also considered, together with the desirable characteristics of a good ventilator which have been studied by a number of authors over the past 20 years. These studies have been prompted by a gradual realisation of the portable mechanical ventilator as an alternative to the BVM in emergency and also by the growing requirement to stockpile small ventilators for use in major emergencies.

## **7.2 Classification of Portable Ventilators**

There are a number of ways that portable ventilators may be classified. The first is using the basic technical specification: This includes:

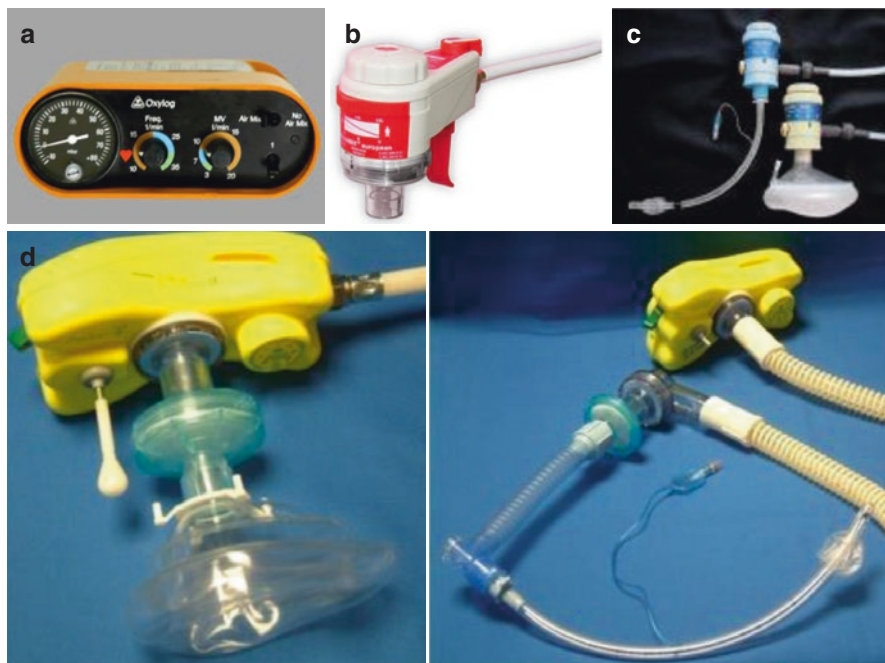
1. Pressure or volume generators—the mode of ventilation provided
2. One two or three control ventilators
3. Pneumatic, electronic-pneumatic or computer-controlled turbine ventilators.

An alternative classification is in terms of what the ventilator is designed to do as follows:

1. Resuscitation ventilators
2. Emergency ventilators
3. Transport ventilators

### **7.2.1 Resuscitation Ventilators**

These devices are designed to be very simple to operate. They are powered by compressed oxygen or air and have only a single control for both tidal volume and frequency of ventilation. Resuscitation ventilators can operate automatically or using a manual override to deliver individual breaths as part of CPR (a cycle of two breaths

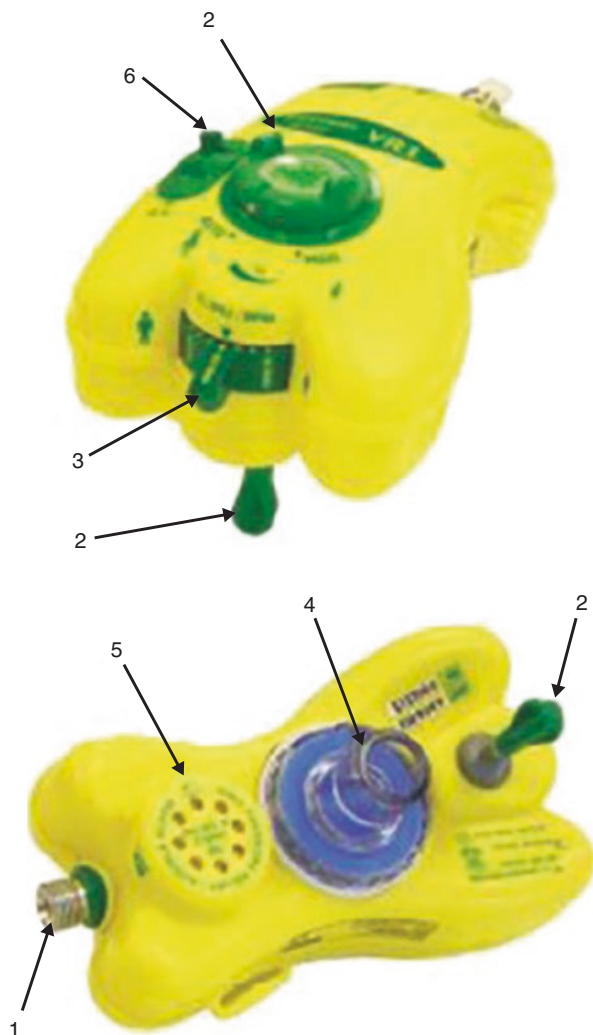


**Fig. 7.1** Examples of resuscitation ventilators (a) Dräger Oxylog (Photograph copyright Dräger SAS, France), (b) Microvent (Photograph courtesy of Microvent, UK) (c) Oxylator (Photograph courtesy of CPR Medical Systems Inc., Canada) (d) the PneuPac VR1 (Photograph courtesy of Smiths Medical International, Luton UK). With the exception of the Oxylator which is a pressure limited device all these ventilators are volume preset flow generators which will deliver a set tidal volume despite changes in airway resistance and lung compliance

followed by 30 chest compressions, according to the ILCOR guidelines). This type of ventilator usually has no monitoring but may be fitted with an audible high pressure alarm to indicate a high pressure in the patient circuit. Figure 7.1 shows some examples.

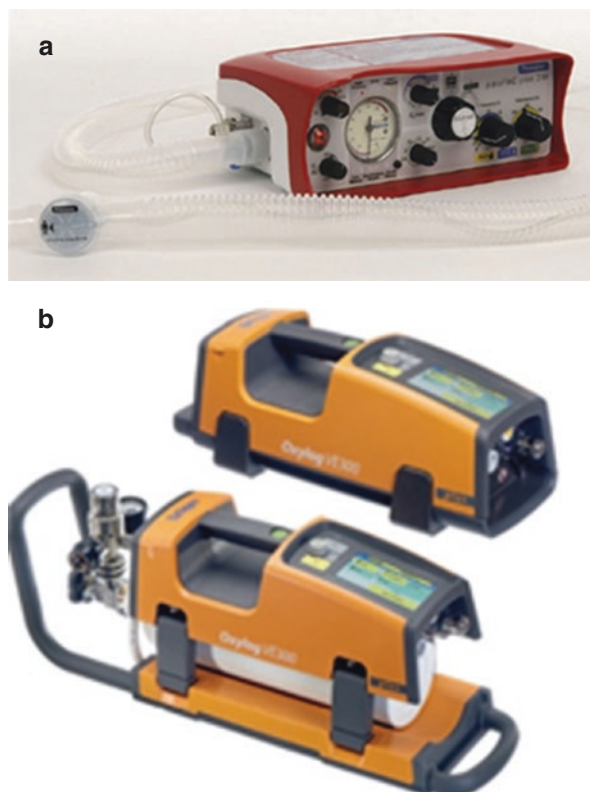
Resuscitation ventilators are designed to deliver ventilation directly to the patient by a pharyngeal mask. This is done through a non-return valve directly attached to the ventilator. In the case of the PneuPac VR1 ventilator (Fig. 7.2) a patient circuit with a second one way valve can be attached to connect with a secure airway such as an endotracheal tube when this is in place. In order to be able to deliver a set tidal volume against variable lung compliance and airway resistance resuscitation ventilators should be time cycled volume preset flow generators (Chap. 6). However, some resuscitation ventilators are pressure cycled which means that they cannot be relied upon to deliver an adequate tidal volume if the lung mechanics are altered. If the airway resistance is high it is possible for such ventilators to cycle from the

**Fig. 7.2** The Pneupac VR1 ventilator (1) driving gas input (2) automatic/manual operation switch and manual release button and lever (3) Vt/f settings control (4) patient valve (5) high patient pressure alarm (6) airmix control (Photograph courtesy of Smiths Medical International, Luton UK)



inspiratory to the expiratory phase without delivering any tidal volume at all. This should be obvious if the patient is being properly observed clinically when the chest will be seen not to rise. When choosing a resuscitation ventilator therefore it is important to find out beforehand how the device operates and what are its limitations (by bench testing against a mechanical test lung before using the device on patients).

**Fig. 7.3** Examples of emergency/transport ventilators (a) Pneupac Parapac Plus 310 (Photograph courtesy of Smiths Medical International, (b) Dräger VN300 (Photograph ©Dräger France SAS,France)



### 7.2.2 Emergency Ventilators

These are portable ventilators with more controls and monitoring devices than a resuscitation ventilator. They consist of a control unit, powered by compressed gas, battery power connected to the patient by a patient circuit and a patient valve. Emergency ventilators are designed to be used for longer term ventilation although they can also be used for resuscitation. They have independent controls for frequency and tidal volume. Monitoring with a manometer shows the inflation pressure during the inspiratory phase and the level of PEEP if applied. In addition emergency ventilators should be equipped with both visual and audible alarms (see Sect. 7.5). These are designed to advise the user of potential problems during ventilation but do not replace the need for constant clinical observation of the patient. Figure 7.3 shows some typical two control emergency ventilators.

### **7.2.3 Transport Ventilators**

Transport ventilators are portable ventilators that are designed, as their name suggests for the transport of patients who require artificial ventilation (but who are stable and are not in an emergency situation) from one location to another. This definition covers a wide span, ranging from patients who have been stabilised at the scene of an emergency and require transportation to hospital to patients who are in specialised intensive care units in hospital who require transportation to another part of the hospital (for example for special investigations such as magnetic resonance imagery) or to another hospital, which may be anything from a few to many thousands of miles away. Transport ventilators are usually more complex than emergency ventilators although there is considerable overlap in design. They range from pneumatic ventilators with more variable controls than the emergency ventilators described above to computer-controlled devices that are in effect miniature versions of ventilators found in the ICU and which are capable of delivering a wide range of ventilation modes. The simplest transport ventilators are designed for use essentially by non-specialists whereas the most sophisticated are used by transport teams that include a specialist anaesthetist or emergency physician. In the United States such a team may include a respiratory therapist but this is a speciality that does not exist in other parts of the world. Figure 7.4 shows a range of advanced transport ventilators. The practice of transport ventilation is a special case of the use of portable ventilators and is covered in the next chapter.

## **7.3 Technical Aspects of Portable Ventilators and Ancillary Equipment**

For many emergency responders who have been trained to use BVM ventilation portable ventilators seem complex and daunting. A device that takes gas from a cylinder at 2300 psi and manages to deliver it to the patient at only 10–15 cm H<sub>2</sub>O (about 0.15 psi) is often treated with suspicion by operators who only ventilate patients occasionally. In order to overcome these doubts this section describes the way high pressure gas is controlled to be able to provide a suitable pressure to ventilate a patient and the function of the essential equipment required.

### **7.3.1 Power Source**

Portable ventilators require a power source to operate. This may be either compressed oxygen, battery power or both. Simple resuscitation, emergency and some transport ventilators are driven only by the power of compressed gas while battery power is required in more complex transport devices, both to drive the ventilator and





**Fig. 7.4** Examples of advanced transport ventilators. (a) Hamilton T1 (Photograph courtesy of Hamilton International, Switzerland) (b) Dräger Oxylog 3000 (Photograph © Dräger France SAS, France) (c) SIEM BA 2001 RA-EL (Photograph courtesy of SIEM, Italy) (d) PneuPac VentiPac 200D (Photograph courtesy of Smiths Medical International Ltd, Luton, UK). With the exception of the VentiPac all these ventilators are power-dependent and use computer control of the delivered ventilation. The VentiPac is pneumatically operated with a wide variety of ventilation settings and an electrically powered monitoring system

to operate the electronic monitoring and control systems. Some ventilators use a battery driven internal compressor to compress the surrounding air as a driving gas. This provides a valuable saving in bottled oxygen when transport is over a long distance and resupply is difficult.

### **7.3.2 *From the Cylinder to the Patient: The Pressure Cascade of Portable Ventilation***

The high pressure of oxygen contained in the cylinder must be reduced in stages and processed through the ventilator before being finally delivered to the patient. The essential stages are as follows:



**Table 7.1** Oxygen cylinder sizes and capacities

Cylinder size	C	D	E	F	G	J
Height (inches/cm)	14/36	18/46	31/79	34/86	49/124	57/145
Capacities (litres)	170	340	680	1360	3400	6800

### 7.3.2.1 Oxygen Cylinders

Compressed oxygen is stored in cylinders of varying sizes at 2300 psi (13,700 kPa). Lightweight modern cylinders made of carbon fibre material can be charged to a higher pressure (about 3000 psi). The contents of the cylinder are described as the volume the high pressure gas inside would occupy at normal atmospheric pressure (about 14 psi or 760 cm Hg). A knowledge of the capacity of oxygen cylinders is important when planning how long emergency and transport ventilation will last and how much compressed gas will be needed (see Chap. 9). Table 7.1 gives details of the sizes of commonly used oxygen cylinders. Sizes above E are usually only found in static oxygen storage facilities in hospitals. The most common form of cylinder is the D size which contains oxygen that would occupy 340 l at atmospheric pressure.

### 7.3.2.2 Reducing Valve

The pressure in the oxygen cylinder is too high to be used directly in connection with a ventilator and has to be reduced to about 40–60 psi before being fed to the ventilator. This is done using a pressure reducing valve which was considered in Sect. 6.5.

### 7.3.2.3 High Pressure Hose and Connector

The reducing valve is clamped to the outlet of the oxygen cylinder. It is connected to the ventilator using a high pressure hose (a pressure of 40–60 psi) which is connected by a Schrader push fit connector. Different types exist depending on whether the ventilator is to be run on compressed oxygen or compressed air which is sometimes the case when oxygen supplies are limited.

### 7.3.2.4 Connection to the Ventilator and Internal Regulation

The high pressure hose is connected to the ventilator usually using a bolted connection. Inside a pneumatically operated ventilator the pressure is further regulated to a standard driving pressure to operate the pneumatic components.

Finally, the portable ventilator delivers gas at a low pressure suitable for ventilating the lungs (usually 10–15 cm H<sub>2</sub>O) via the patient circuit described in Chap. 6 at a tidal volume, pressure and frequency determined by the controls on the ventilator.

## 7.4 The Basic Controls of a Portable Ventilator

The controls on a portable ventilator vary according to the manufacturer, the type of ventilation being delivered and the complexity of the ventilator. In general the controls become more complex between basic resuscitation ventilators, emergency ventilators and ventilators designed for patient transport after stabilisation in hospital. Despite this a number of controls are common to all ventilators. These are:

### 7.4.1 Tidal Volume

This calibrated control sets the tidal volume delivered to the patient circuit from the ventilator. Note that this is not necessarily the tidal volume that is delivered to the patient, which may be lower due to leaks in the circuit. The actual tidal volume delivered is equal to the expired tidal volume. This can be measured on more complex models of portable ventilator with a Y patient circuit or by inserting a spirometer between the patient valve and the ETT.

The default tidal volume setting on portable ventilators is usually 600 ml to conform to the ILCOR guidelines. One important point about tidal volume on portable gas-powered devices is that the calibration is only approximate and depends on the frequency of ventilation. This is because the control is actually regulating flow of gas through a needle valve in the ventilator circuit and not measuring a set amount of gas by volume. In practice, for portable gas powered ventilators it is possible to deliver a range of tidal volumes over a range of ventilation frequencies to achieve the correct minute volume, but in practice only a limited number of V<sub>t</sub> and frequency combinations will deliver the tidal volume indicated on the control. Figure 7.5 shows details from a Pneupac Parapac ventilator. On this particular ventilator colour coding is used between the V<sub>t</sub> and frequency controls to aid the operator in setting the right combination. Not all ventilator manufacturers make this clear however. It is therefore important to bench test any ventilator during commissioning before it is used on patients to determine the accuracy of the V<sub>t</sub> control.

Calibration of Tidal Volume Control

Actual tidal volume in millilitres delivered at combinations of BPM & Vt knob settings									
Set Tidal volume ► Set Frequency ▼	1500	1000	700	500	350	200	150	100	Min
8	1500	1235							
10	1215	1000	832						
12		842	700	619					
15			566	500	458				
20				383	350	255			
25					275	200	171		
30						175	150	108	82
40							139	92	70

Actual Frequencies are within  $\pm 20\%$

Actual 100% FIO<sub>2</sub> Tidal volumes are within  $\pm 20\%$  or  $\pm 50\text{mL}$  whichever is the greater

Actual 50% FIO<sub>2</sub> Tidal volumes are within  $\pm 25\%$  or  $\pm 50\text{mL}$  whichever is the greater

All tidal volumes are referenced to Standard Temperature and Pressure (STP) i.e.

1013mbar and 21°C for a normal lung of compliance of 0.05 L/cm H<sub>2</sub>O (C50) and resistance 5cmH<sub>2</sub>O/L/sec (Rp 5).

**Fig. 7.5** Delivered tidal volume vs frequency settings of the Pneupac Parapac Plus 310 ventilator (Reproduced with permission, Pneupac Ventilation, Smiths Medical International, Luton, United Kingdom)

### 7.4.2 Frequency Control

This controls the number of breaths delivered to the patient per minute. In portable gas-powered ventilators the control operates a pneumatic timing device such as the pneumatic oscillator. The flow of gas in and out of this spring-loaded device determines the rate of delivery of ventilations. The actual rate required depends on the age of the patient. Children require higher rates than adults. The normal frequency range provided on most portable ventilators is between 8 and 40/min. For adults the default ventilation rate recommended by ILCOR is 12/min.

### 7.4.3 Combined Tidal Volume and Frequency Controls

In basic resuscitation ventilators there is one control which controls both tidal volume and frequency. The range of settings is from child setting, with a low Vt and high frequency through to adult settings with a higher tidal volume and lower frequency (Fig. 7.6).

**Fig. 7.6** Manual ventilation button and the Vt/f control on the Pneupac VR1 resuscitation ventilator (Photograph courtesy of Pneupac Ventilation, Smiths Medical International, Luton, United Kingdom)



#### **7.4.4 I:E Ratio**

The ratio of inspiratory to expiratory times in a portable ventilator is usually set to 1:2 with the internal timing device. However this value can be extremely variable depending on the model used, the settings of frequency and tidal volume. Almost all portable gas-powered ventilators have variations in I:E ratio across the range of delivered ventilation and these should be indicated by the manufacture in the user's manual. Before buying a ventilator therefore it is important to check that this information is provided and that the limitations of the ventilator at different settings are understood.

##### **7.4.4.1 Variable I:E Ratio**

For non-specialist ventilator users the fixed value of 1:2 is recommended. However in certain conditions a variable I:E ratio is useful, for example in the management of severe bronchial asthma where the expiratory time is extended. Variation of I:E is standard practice in ICU ventilation. Variable I:E is provided on some portable

ventilators by having separate controls for the timing of the inspiratory and expiratory phases. This means that a wide range of I:E settings is possible.

### **7.4.5 *On/off and CMV/Demand Controls***

#### **7.4.5.1 Manual or Automatic Ventilation**

When a portable gas - powered ventilator (PGPV) is switched on the ventilator cycles at the set rate and tidal volume. This automatic controlled mandatory ventilation can be overridden by the demand valve which detects whether the patient is breathing adequately. This mode of ventilation is described as Controlled Mandatory Ventilation (CMV) /Demand setting in ventilators where there is a demand valve fitted. This form of control means that automatic ventilation will be delivered unless the patient shows adequate ventilation efforts and is able to suppress the ventilator.

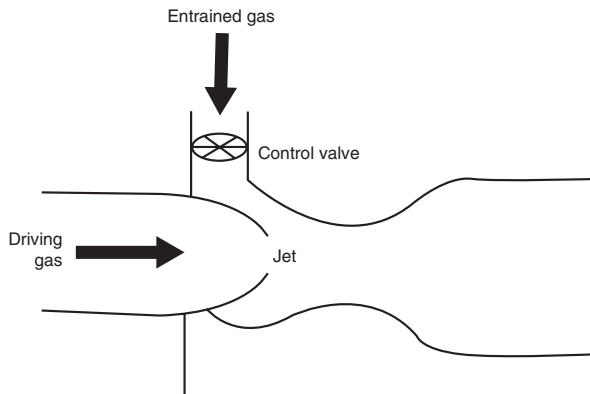
If the ventilator is set to 'Demand' (or 'Off' in earlier PGPV), the patient has been seen to be breathing adequately and when taking a breath, the ventilator delivers oxygen at atmospheric pressure during the inspiratory phase. This is an efficient way of delivering oxygen therapy, compared with constant flow through a face mask where the oxygen flow is delivered during both the inspiratory and expiratory phases. Note that the delivered oxygen in the 'Demand' setting of a PGPV is at atmospheric pressure. It does not therefore provide pressure support ventilation (Chap. 6).

#### **7.4.5.2 Manual Override**

Following the changes in the ILCOR CPR recommendations in 2010, re-endorsed in 2015 that two breaths should be delivered following 30 chest compressions a number of manufacturers have included a manual override system into resuscitation and emergency ventilators. This means that when the ventilator is switched to the 'Off' setting it is possible to deliver manually controlled ventilations which conform to the standards recommended by ILCOR. Figure 7.6 shows the manual release system on the Pneupac VR1 ventilator which allows the delivery of individual controlled breaths. In the case of this ventilator, the breath delivered is exactly the same as that which would be delivered if the ventilator were operating automatically. In other words it is delivering one machine controlled ventilation. If the manually delivered ventilation is interrupted, the next ventilation will deliver what is remaining from the first.

It should be noted that this system of ventilation is not the same as a manual insufflator where gas flows continuously into the patient until the trigger is released.

**Fig. 7.7** The Venturi principle of entraining gas



Again, when selecting a ventilator it is important to understand what is being delivered when manual ventilation is selected.

#### 7.4.6 Airmix Control

Portable ventilators that are powered by compressed oxygen deliver a fixed concentration of 100% to the patient. This is appropriate for emergency ventilation for life-threatening hypoxia where the  $FiO_2$  should be as high as possible, as given by the alveolar air equation. However, the concentration of inspired oxygen should be reduced as soon as possible while still providing an adequate  $PaO_2$  to avoid problems of oxygen toxicity. In CPR the ILCOR guidelines recommend that inspired oxygen concentration should be reduced after the initial hypoxia has been resolved, since this has been shown to improve long term survival from cardiac arrest. Another reason for reducing the inspired oxygen concentration is to prolong the life of the cylinder providing the gas.

Therefore on many portable gas-powered ventilators there is an airmix control which reduces the delivered oxygen concentration to between 40 and 50%. As its name suggests airmix mixes ambient air with oxygen fed through a curved tube called a venturi (Fig. 7.7). When a flow of gas is passed through a venturi it creates a partial vacuum which draws air in and mixes with the oxygen to provide a lower delivered oxygen concentration. The actual level achieved depends on the flow of oxygen which is sucking in air containing 21% oxygen.

The performance of airmix systems can be very variable depending on the tidal volume and frequency settings of the ventilator and the compliance and resistance of the lungs being ventilated. Therefore when using an airmix system the oxygen saturation of capillary blood should be monitored using a pulse oximeter and blood gases if possible for longer term ventilation.

Airmix should not be used where there is a possibility of contamination, either chemical or biological (bacterial and viral) in the surrounding atmosphere.

### **7.4.7 Blow-Off Pressure Control**

All portable ventilators must have a built-in pressure relief valve to prevent over-ventilation of the lungs. This is particularly important with volume preset flow generators where the inspired pressure rises until the desired tidal volume has been delivered. If the lung compliance is low and the airway resistance high, the pressure can rise to high levels which could cause damage to the lung tissue. Therefore a pressure relief valve is included in the part of the ventilator delivering gas to the patient. This is usually fixed at a preset value of 40 cm H<sub>2</sub>O (or 60 cm in the USA). However, many portable ventilators allow the operator to set the blow off pressure at any value up to 60 cm. For the non-specialist a value of 40 cm is recommended. The setting can be checked by switching the ventilator on and occluding the patient circuit. The pressure on the manometer (Sect. 7.5.2.5) if fitted should be seen to rise up to 40 cm when the pressure will be released. This is usually accompanied by an audible high pressure alarm.

When selecting a ventilator it is important to find out how the high pressure relief system operates from the manufacturer's information. Some ventilators release all the gas from the inspiratory phase to the atmosphere when the preset high pressure point is reached. This means that none of the gas goes into the patient during the inspiratory phase. However, some ventilators such as the Pneupac Parapac Plus feed all the gas that has been delivered up to the high pressure relief point into the patient while releasing only the gas that is delivered after the high pressure point has been reached to the atmosphere. This ensures that some ventilation of the lungs takes place.

Sudden action of the high pressure relief valve usually indicates that the patient circuit is blocked or kinked from the outside. High pressure relief when preceded by a gradual increase in inspiratory pressure shown on the manometer indicates a problem related to the lung mechanics rather than to the ventilator and its patient circuit.

## **7.5 Monitoring on Portable Ventilators**

### **7.5.1 Introduction**

Portable ventilators have a variable number of alarms and monitoring facilities that are provided to alert the operator to any potential failure of artificial ventilation, whether related to the patient's lung and airway or to the function of the ventilator itself. Certain basic alarms are common to all classes of portable ventilator but with

**Fig. 7.8** The Pneupac Parapac Plus 310 emergency and transport ventilator showing controls and alarms (Photograph courtesy of Pneupac Ventilation, Smiths Medical International, Luton, United Kingdom)



the increasing sophistication of the high end transport ventilators the monitoring too becomes more complex, providing the user with the type of information expected from a modern hospital ventilator.

Whatever the level of monitoring and alarms provided on a ventilator the essential point is that these can only aid and not replace good clinical practice. When a patient is being ventilated in emergency and during transport he or she must be observed clinically at all times. This means both visual and auditory assessment as described in Chap. 4. The colour of the patient and the rise and fall of the chest in synchrony with the ventilator are basic and essential features of clinical monitoring. No patient who is being ventilated in emergency or during transport should ever be left unattended. While the ventilator operator may be required to help with other tasks during emergency the ventilation should always take priority. However, to assist the ventilator operator with this task and to help assess the effectiveness of artificial ventilation the provision of alarms and other monitoring on the ventilator provides very useful clinical support.

As with clinical observation of the ventilated patient ventilator monitoring may be conveniently be divided between visual and audible monitoring and alarms.

### 7.5.2 Visual Monitoring and Alarms

The type of visual monitoring provided on a portable ventilator will depend on the degree of complexity of the device and how it is powered. Many modern devices depend not only on compressed gas but also a battery to operate the ventilator. This allows an increased degree of sophistication in the visual monitoring but comes with the disadvantage that the useful endurance of the ventilator is limited by the battery life. This can be overcome when the ventilator is being used in an environment such as an ambulance where an alternative source of power is available. The actual visual monitoring provided on any portable ventilator will depend on the model in question. The following essential visual monitors and alarms are common to many models. The reader is advised to check exactly what is available and how it functions on any ventilator before making a purchase.



### 7.5.2.1 High Pressure Gas Supply

The cylinder used to power a PGPV will have a pressure gauge built into the regulator/reducing valve which gives an indication of the pressure of the gas remaining in the cylinders. In addition, PGPVs are provided with a pneumatically-operated fish eye indicator, which shows (1) whether the driving gas is turned on at the cylinder control and (2) whether the gas pressure to the ventilator is sufficient to drive it. The fish-eye high pressure indicator is also linked to an audible alarm which sounds when the pressure of the gas driving the ventilator is inadequate (see below). The fish eye indicator shown in Fig. 7.8 is a typical example. A white shutter inside the fish eye indicates adequate gas pressure while a red shutter indicates inadequate pressure. The fish eye indicator is completely pneumatic in operation and does not involve any electronics. Most emergency ventilators also have a number of electronic alarms which may operate as part of the ventilator itself or as an independent battery-powered monitoring system. These alarms are indicated by light-emitting diodes (LED). A typical array of LED alarms built around a manometer is also shown in Fig. 7.8.

### 7.5.2.2 High Patient Inspiratory Pressure Alarm

This alarm shows when the peak pressure in the patient circuit during inspiration exceeds the limit set by the blow-off valve described previously. This will be either 40 or 60 cm with a fixed relief valve or at any level set by the operator above the established peak inspiration pressure. The visual alarm is also accompanied by an audible alarm which sounds at a standard high priority. The high pressure alarm is important in detection of a blocked airway (e.g. a kinked ETT or secretions inside) and also blockage of the patient circuit (a common cause is the circuit being kinked while loading the patient into an ambulance or any other situation where a patient on a ventilator is being moved from one position or location to another. This is a time in AV when clinical monitoring should be at its most vigilant).

### 7.5.2.3 Low Patient Inspiratory Pressure Alarm

This LED alarm, which is also accompanied by a characteristic high priority audible alarm indicates that the pressure in the patient circuit has fallen during inspiration to a level which will not ventilate the patient. Causes of this are usually disconnection of the ventilator from the patient airway or displacement of the ETT or other airway device. Any leak in the patient circuit will activate the low pressure alarm. This alarm is usually set by the manufacturer at 10 cm H<sub>2</sub>O.

#### 7.5.2.4 Patient Triggering Indicator

This LED alarm indicates when a patient is taking a spontaneous breath which is sufficient to suppress the automatic action of the ventilator and move over to demand mode. This will occur if the breath taken is sufficient to suppress the ventilator through either SIMV or SMMV.

#### 7.5.2.5 Manometer

A manometer is a device which measures the pressure in ventilator and patient circuit during both the inspiratory and expiratory phases. A typical example is shown in Fig. 7.8. During the inspiratory phase the pressure is usually between 10 and 20 cm H<sub>2</sub>O. This pressure will rise to a maximum in a ventilator that is a time-cycled flow-generator (Sect. 6.4.2) or will immediately rise to a preset pressure on a pressure generating device. Remember in this case that the indication of the positive airway pressure does not necessarily indicate flow into the lungs. Only clinical observation of the rise and fall of the chest supplemented by measurement of tidal volume can confirm that gas is entering and leaving the lungs. The manometer is calibrated to show high inflation pressures in red.

During expiration the manometer pressure will fall to almost zero unless PEEP has been set. If this is the case on the ventilator being used then the PEEP level will be indicated on the manometer. Note that most portable ventilators do not always release all of the expired gas from the lungs to the atmosphere. This is due to design constraints. The PEEP remaining in the patient circuit is usually only small (of the order of 2–3 cm H<sub>2</sub>O) and is called intrinsic or auto-PEEP. When CPAP is being used on some ventilators this too will be indicated on the manometer.

#### 7.5.2.6 Battery Indicator

On portable ventilators that are dependent on a battery for their function a battery indicator should be provided to indicate both the existing battery power and the approximate duration of the remaining charge.

### 7.6 Audible Alarms

On pneumatic ventilators audible alarms accompany the visual alarms as noted above. They are grouped according to an agreed international priority system where high frequency notes are issued according to the alarm type. The low driving gas alarm is a continuous tone which is distinct from the other alarms.

## **7.7 Using a Portable Ventilator: Essential Checks Before Starting Ventilation**

### **7.7.1 Introduction**

Before using any portable ventilator the maker's handbook should be studied in detail to find out the features of the device, its controls, the type of ventilation being delivered and particularly the alarm system. There are specific checks to be carried out before using the device clinically for the first time. Experience should be gained during training using the various controls with the ventilator connected to an artificial lung (or more simply an anaesthetic Boyle bag). There are a number of artificial lung systems available on the market which can provide a range of settings of lung compliance and airway resistance. A typical model was described in Sect. 5.6.6. The great advantage of thoroughly testing a ventilator over a range of compliance and resistance settings is that the limitations of any particular ventilator will be revealed, for example variations in delivered tidal volume. Often the manufacturer's handbook will provide this information but this is not always the case.

### **7.7.2 Operator's Handbook**

The handbook for any ventilator will give the standard checks that should be carried out before use, both generally as in the case of starting a shift on an ambulance or specifically when the ventilator is being used on a patient. The startup checks vary according to the ventilator and it is important that the user should be familiar with the checks for the device he is using. Checks should preferably be carried out reading from the handbook, following the practice of pre-take off checks on an aircraft and modern practice in checking anaesthetic equipment.

The following sections are taken from the user's manual for the Pneupac Parapac Plus emergency ventilator which is given as a training example.

#### **7.7.2.1 Pneumatic Power Checks**

1. Check the cylinder oxygen supply and regulator. Is the pressure gauge showing adequate contents of compressed oxygen. Will the contents be sufficient to operate the ventilator during the time foreseen for the emergency response. This depends on the setting of the emergency. The cylinder should preferably be kept full.
2. Check that a cylinder key is available to open the cylinder. This should ideally be attached to the regulator by a chain so that it cannot be removed.

3. Check that the cylinder valve opens and that there is no defect in the thread. Check that oxygen flows from the regulator if possible.
4. Connect the ventilator to the reducing valve using the click in Schrader connector.
5. Check that the fish eye indicator of oxygen supply has turned from red to white.
6. Turn off the oxygen supply until the ventilator controls are ready for checking.

### 7.7.2.2 Ventilator Controls

1. Check the ventilator controls are set as follows:
  - Main Pneumatic Switch: 'Demand' or 'Off'
  - Frequency: 12 b/min (default position)
  - Tidal Volume: 600 ml
  - Air Mix Switch: 'No Air Mix'
  - Relief Pressure:  $40 \times 100$  Pa (40 cmH<sub>2</sub>O)
  - (this value is fixed as a default but can be operator varied in some models)
2. Connect the probe on the input hose to the cylinder and reducing valve or central high pressure oxygen source if the ventilator is used in a hospital setting.
3. If connected to a cylinder regulator turn on cylinder valve *slowly*.

NOTE: The gas source must be capable of maintaining a pressure of at least 305 kPa ( $\approx 3$  bar) whilst delivering a flow of 65 L/min.
4. Check that the visual alarm for supply gas failure has *changed from red to white*.
5. Connect the ventilator to the patient circuits and inspect the patient valve for the presence of any foreign bodies (e.g. packing materials) that could impair function.
6. Switch the main pneumatic switch to 'CMV/Demand' or 'On.' The ventilator should commence cycling and all the alarm lights flash in turn. A single burst of the high priority audible alarm is given at the same time. The orange silencing indicator should flash for 60 s. Check that the flow is coming from the patient connection port by feeling the flow when placed close to the back of the hand or to the face.
7. Occlude the output port on the patient valve with the palm of the hand and check that the manometer gives a reading of between 30 and 50 cm during each inspiratory phase. The pneumatic audible alarm should also sound, accompanied by the high inflation pressure visual alarm. After occlusion for one second, once the silencing period has elapsed, the high priority electronic audible alarm will also sound. Check that the unit cycles regularly about every 5s.
8. Switch over to 'Air Mix' and repeat step 6. The change in the manometer reading should not exceed  $5 \times 100$  Pa (100 Pa = 1 cm H<sub>2</sub>O).

NOTE: After the 60s initial silenced period the electronic audible alarms will operate if an alarm condition persists. These can be silenced for as long as

required by depressing the silencing button each time the silencing indicator switches off.

9. Set the 'Tidal Volume' control to its minimum setting. Occlude the output port and check that a pressure of at least  $20 \times 100$  Pa is attained on the manometer. Gradually increase the flow setting and observe how the pressure rises—demonstrating the pressure generator principle. At the end of the green segment the pressure should be attaining the nominal set value.
10. Reset the 'Tidal Volume' control to its minimum setting and select 'No Air Mix'. Occlusion of the output port should now cause the manometer to rise sharply to between  $30$  and  $50 \times 100$  Pa and the alarms should operate.
11. Allow the ventilator to cycle with no obstruction at the output port and check that the low inflation pressure (disconnect) alarm operates after 10s.
12. Set the 'Frequency' and 'Tidal Volume' control knobs to the extremes of their range. By listening to the gas flow, check that the ventilator is responding to the controls and that no irregularities of performance can be discerned.
13. If the ventilator is likely to be used with infants and children the following additional test should be carried out during the periodic test and after reassembly of the patient valve every time it is dismantled:  
Connect the ventilator to a gas source and set the 'Tidal Volume' control to minimum and the air mix switch to 'Air Mix'. Attach a flexible reservoir bag (preferably 0.5 l) to the patient connector of the patient valve and switch on the main pneumatic switch. Roll up the end of the reservoir bag to decrease its effective volume until the end inspiration inflation pressure rises to about  $10 \times 100$  Pa. Check that this pressure can be attained consistently every breath. If it cannot, dismantle the patient valve, turn the yellow valve element about a quarter of a turn and reassemble and retest. If after two or three adjustments consistent performance cannot be achieved the valve element must be replaced.
14. Finally, set the controls as specified in step 1 so that the ventilator is left set for ventilation at normal adult settings.

## **7.8 Using a Portable Ventilator in an Emergency: Practical Steps**

### **7.8.1 Introduction**

There can be no substitute for hands-on training in using a portable ventilator both with simulators or advanced manikins such as the Laerdal Sim-Man™ and on patients who are not in an emergency but in the controlled circumstances of the operating theatre, where much of non-specialist airway and ventilation training is

carried out. The following notes are therefore only a guide and an aide memoire for the practice of artificial ventilation using a portable ventilator. Many manufacturers give appropriate notes for the use of their own devices in the handbook and the operator is again advised to read these carefully.

A *key rule* that is appropriate to all portable ventilators however is that an alternative ventilation device, such as a BVM should *always* be available to take over ventilation when there is an unexplained failure to ventilate.

### 7.8.2 *Commencing Artificial Ventilation*

The ventilator should always be left with the controls set in the position specified in the functional checks described above to enable it to be brought into use with a minimum of re-adjustment. It should be stored with a suitable gas source or suitable wall outlets must be known to be available. At least one patient circuit should also be kept available for emergency use.

1. Connect supply hose probe to dry filtered gas supply.
2. Turn on gas supply (if relevant).
3. Check that the visual alarm for supply gas failure has changed from red to white.
4. Turn Function switch to CMV.
5. Check that the alarm indicators flash in sequence, to indicate correct function.
6. Set ventilation parameters to suit the patient.
7. Briefly occlude the patient connection port of the patient valve with the thumb. Check that the peak inflation pressure reading on the manometer is appropriate for the patient and that the pneumatic audible alarm sounds and the high inflation pressure indicator shows red.
8. Connect face mask or endotracheal tube (ET tube) to the patient valve.
9. Check chest movement and inflation pressure manometer to ensure correct ventilation.
10. Check that the green cycle indicator light flashes when inflation pressure rises above 10 cmH<sub>2</sub>O.
11. Make adjustments to the ventilator based upon clinical observation and measurement of expired CO<sub>2</sub> and pulse oximetry.

The patient's condition and chest movements as well as the inflation pressure monitor should be kept under constant observation so that changes in airway resistance and lung compliance can be detected and corrected before the patient is put at risk. Repeat step 11, above, if the tidal volume setting is increased at any time during the ventilation procedure. When ventilating with a mask, the peak inflation

pressure should ideally be kept below 20 cmH<sub>2</sub>O (20 × 100 Pa) to minimise the risk of inflation of the stomach.

If the pressure jumps excessively at the commencement of inspiration, an airway obstruction is indicated and this must be rectified. If the airway is clear the flow rate may be too high and this should be reduced by decreasing the tidal volume setting.

Excessive pressure at the end of inspiration may be the result of a too high setting of tidal volume. This may be reduced by either reducing the tidal volume setting or increasing the frequency setting.

When ventilating with an ET tube a sudden increase in patient airway pressure may indicate kinking of the tube or other obstruction.

If the inflation pressure is too low, particularly if the low pressure alarm operates;

1. check for leaks
2. check the ventilation parameters
3. check the patient valve for proper functioning

## 7.9 Using Portable Ventilators in Emergencies: The Evidence Base

The previous chapter considered some of the disadvantages of the use of the BVM device in emergency ventilation. Although these devices are widely used around the world there is increasing recognition of their limitations. The alternative to using a BV device in emergency is the portable automatic ventilator. Portable mechanical ventilators have a number of advantages over bag-valve devices however they have not been studied as intensively as other methods of providing artificial ventilation. The International Liaison Committee on Resuscitation (ILCOR) reviewed the relatively limited literature about automatic ventilators in 2010. The observations about them are reproduced in Box 7.1.

### **Box 7.1 International Liaison Committee on Resuscitation Assessment of Portable Automatic Ventilators (2010)**

1. Automatic ventilators or resuscitators provide a constant flow of gas to the patient during inspiration; the volume delivered is dependent on the inspiratory time (a longer time provides a greater tidal volume). Because pressure in the airway rises during inspiration, these devices are often pressure limited to protect the lungs against barotrauma.
2. An automatic ventilator can be used with either a facemask or other airway device (e.g., tracheal tube, supraglottic airway device).

3. An automatic resuscitator should be set initially to deliver a tidal volume of  $6\text{--}7\text{ ml kg}^{-1}$  at  $10\text{ breaths min}^{-1}$ . Some ventilators have coordinated markings on the controls to facilitate easy and rapid adjustment for patients of different sizes, and others are capable of sophisticated variation in respiratory parameters. In the presence of a spontaneous circulation, the correct setting will be determined by analysis of the patient's arterial blood gases.
4. Automatic resuscitators provide many advantages over alternative methods of ventilation.
  - a. In unintubated patients, the rescuer has both hands free for mask and airway alignment.
  - b. Cricoid pressure can be applied with one hand while the other seals the mask on the face.
  - c. In intubated patients they free the rescuer for other tasks.
  - d. Once set, they provide a constant tidal volume, respiratory rate and minute ventilation; thus, they may help to avoid excessive ventilation.
  - e. They are associated with lower peak airway pressures than manual ventilation, which reduces intrathoracic pressure and facilitates improved venous return and subsequent cardiac output.
  - f. A manikin study of simulated cardiac arrest and a study involving fire-fighters ventilating the lungs of anaesthetised patients both showed a significant decrease in gastric inflation with manually triggered flow-limited oxygen-powered resuscitators and mask compared with a BVM.

### ***7.9.1 The Need for More Studies on Automatic Ventilators***

It is worth noting that the 2010 ILCOR guidelines cited 23 papers which studied ventilation as opposed to more than 93 papers concerned with airway management. Of the ventilation papers cited, only three directly concerned the use of automatic ventilators. The reason for there being so few studies on manual and automatic ventilation is unclear but may be as a result of ventilation being seen as an 'obvious' technique which does not require investigation. In this respect it is similar to suction aspiration which is an essential part of clearing the airway of secretions and vomitus but which appears never to have been the subject of a controlled trial. The evidence available shows that the use of bag-valve devices is associated with hyperventilation, in terms of both frequency and delivered tidal and minute volumes, as well as the peak airway pressure delivered. The consequences of this in terms of possible gastric insufflation and barotrauma are understood. The potential dangers from volutrauma however has received less attention, although the damage caused to the lung parenchyma and subsequent acute respiratory distress syndrome (ARDS) in the intensive care setting have been recognized for many years and have led to the use of smaller tidal volumes with PEEP to keep the alveoli open, (the 'open lung'



strategy first described by Lachmann (see Chap. 11)). Given the potential vulnerability of the lungs and other organs in a patient with major trauma and shock, the question of induced volutrauma in emergency ventilation deserves greater study.

### ***7.9.2 Bag-Valve Ventilation Compared with Automatic Ventilation***

There have been some studies which compare the quality of ventilation delivered by portable automatic ventilators (known as ‘automatic transport ventilators’ in the US, despite the fact that they are widely used in emergency ventilation as well as in the transport of a ventilator-dependent patient from one location to another).

Salas et al. (2007) found no differences in delivered tidal volume between a bag a valve device and an automatic ventilator (Impact 730) using a facemask in a model of adult cardiac arrest. However, as noted above they also found less gastric insufflation and a reduced mask leak using the ventilator. Their overall conclusion was ‘that compared with the BVM the ventilator is at least as effective, is easier to use, and limits gastric insufflation.

Weiss et al. (2005) in a study of paramedical personnel using a BVM or an automatic ventilator in cardiac arrest concluded that they were able to accomplish more tasks and provide better patient care when using the automatic ventilator. Goedeke et al. (2006) compared ventilation with a bag valve device and the Oxylator ventilator. They found the bag valve device delivered higher peak airway pressures and was associated with a lower SaO<sub>2</sub>. They found no differences in the tidal volumes delivered.

In another study, comparing the Oxylator resuscitator with bag valve ventilation Noordergraaf et al. (2004a, b) found that the bag valve device was associated with better airway management and that the Oxylator in automatic mode delivered hyperventilation. They recommended that the ventilator should only be used in manual mode during resuscitation.

The function of the automatic ventilator is important (whether the device is a pressure or a volume generator) in replacing BV ventilation. Pressure cycled ventilators have been shown to be unreliable in emergency use. L’Her and Roy (2011) have conducted laboratory trials of a number of portable ventilators. They concluded that while most of the volume-cycled ventilators proved to be technically efficient and reliable, pressure cycled ventilators (they examined the Oxylator EMX and the Vortran RTM) gave rise to concern since they did not deliver consistent tidal volumes and under certain conditions could be unsafe. Further comment on this is found in a review by Branson (2011).

## 7.10 Conclusions

1. Portable mechanical ventilators for use in emergency and in the transport of ventilator-dependent patients have been developed in parallel with hospital ventilators over the past 40 years. Many such devices exist today ranging from small, hand-held devices for use in resuscitation through to complex computer-controlled transport ventilators that have most of the features of a modern intensive care ventilator.
2. Portable ventilators may be classified by the type of artificial ventilation they generate (either pressure or volume generators), the number of basic controls with which they are equipped and by their power source (electrical or pneumatic).
3. An alternative classification is as resuscitation, emergency and transport ventilators.
4. A basic knowledge of the operation, controls and alarms of a portable ventilator is essential for its correct use.
5. For pneumatic ventilators there is a cascade of reduction of oxygen pressure from the driving cylinder through to the patient's airway via the reducing and regulator valves, the ventilator itself and the patient circuit.
6. Monitoring of the performance of a portable ventilation is provided in emergency and transport ventilators. Monitoring of cycling and inflation pressure is accompanied by visual and auditory alarms to alert the operator to a possible malfunction of artificial ventilation.
7. Standard checks specified by the manufacturer should be carried out by the user before putting a new ventilator into service and before each use.
8. A new ventilator should preferably be tested against a calibrated test lung to check the calibration and whether or not the ventilator will actually deliver according to the manufacturers' claims.
9. The practical use of a portable ventilator should be familiar to operators through training and ventilation of manikins before working with patients. A flight-style check list before each use of a ventilator is helpful.
10. The clinical evidence base for the use of portable ventilators in emergency and transport is relatively limited compared with the study of airway management. There is a need for more work in this area. A number of clinical studies have been published comparing the bag-valve mask with portable mechanical ventilation. These have shown the potential deficiencies of the BVM and advantages of mechanical ventilation.

## Suggestions for Further Reading

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## Chapter 8

# Managing Ventilation During Transport



### 8.1 Introduction

Previous chapters have considered the provision of artificial ventilation in an emergency to overcome life-threatening hypoxia. Transport ventilation is different from emergency ventilation and concerns the safe movement of a patient who is dependent on a ventilator but in a stable condition from one location to another. Broadly this covers three situations:

1. transport from the site of an emergency to the hospital
2. transport within the hospital (intrahospital transport)
3. transport between hospitals

These three settings cover distances that may be only a few metres, to thousands of kilometres. Distance is not the only consideration however. In the hospital setting for example, transport times may be very short (for example moving the patient from the operating theatre to the ICU) or several hours (when a patient requires lengthy diagnostic or therapeutic procedures).

Although transport ventilation is different from emergency ventilation, there is considerable overlap between the two. In emergency ventilation the objective is to correct hypoxia and stabilise the patient as quickly as possible. In transport ventilation the objective is to move a patient who is being ventilated and who is in a critical but stable situation safely from one location to another with no reduction in the quality of ventilation provided, nor the clinical condition. To provide safe and effective transport ventilation requires good clinical skills and judgement along with suitable and effective equipment.

There are inherent risks during transport ventilation associated with both the location and the movement of the patient. Whereas patients being ventilated in the ICU in hospital are in surroundings that are completely adapted to the provision of artificial ventilation by often sophisticated ventilators (e.g. ICU, the operating

theatre or the emergency room) patients being transported and ventilated are in settings that are quite different, for example in the radiology department.

In order to minimise transport ventilation risks, careful planning, training and the provision of appropriate equipment that is familiar to its users is necessary. The past 20 years have seen the development of a wide range of portable ventilators which are used in transport ventilation by medical personnel with varying skill levels. The modern development of ventilators designed for transport has led to the production of devices that are effectively miniature versions of the most complex ICU ventilators which are now in regular use around the world (see Chap. 11). Equally, there are many simpler types of portable ventilator which can be used in transport ventilation by non-specialist medical personnel.

Transport of patients from one ICU to another and in most intrahospital transfers is usually controlled by specialists in the area, including intensive care physicians and nurses and in the United States, respiratory therapists. This chapter considers transport ventilation by non-specialists within the scope of this book where simpler forms of ventilator will normally be used. This may be the case in transport of the patient to hospital in emergency and also within the ER itself.

However, it is important that non-specialists providing basic transport ventilation should have an understanding of specialist transport ventilation and the function of more sophisticated ventilators that are now available for patients whose condition may be controlled only by the type of ventilation modes available in the ICU. The essential problems of transport ventilation are common to both.

## 8.2 Transport Ventilation by Non-Specialists

Non-specialists involved in ventilation will include emergency physicians, nursing and paramedical staff. Medical personnel working in remote locations (Chap. 10) and military medical personnel should also be included.

Interhospital transport of ventilator-dependent patients in the ICU is common in developed countries. It is operated by specialist teams and is not within the scope of this discussion. However, it is helpful for the non-specialist to have an understanding of what transport ventilation involves and the potential problems.

Situations where the non-specialist will be involved in transport ventilation are summarised in Box 8.1.

### **Box 8.1 Situations Where Non-specialists May Be Required to Provide Ventilation During Transport**

- Following stabilisation of a respiratory emergency and subsequent transport to a hospital emergency service
- During transfer from the ambulance to the ER

- Moving the patient during the initial assessment in the ER
- Moving the patient from the ER to imaging units, including MRI
- As part of a specialist–lead team moving a ventilator–dependent patient from the ICU to the operating theatre or to imaging units
- During inter-hospital transportation of patients in remote and other locations where specialist experience may not be available

## 8.3 Managing Transport Ventilation

### 8.3.1 *Guidelines and Training*

The foundation of good transport ventilation is adherence to guidelines, constant checking and monitoring and being aware of the potential problems that can affect any patient who is being moved. During any movement there is a risk that the airway may be compromised along with the connection to the patient. In a cramped environment such as an ambulance or helicopter this is a particular risk. Recognising this, guidelines for the conduct of transport ventilation have been produced (Guidelines Committee ACoCCM Society of Critical Care Medicine and Transfer Guidelines Task Force 1993). These address the requirements for pre-transport co-ordination and planning and communication. The guidelines relate to:

1. Personnel—who should be involved? What should be their qualifications and skills?
2. Equipment—how should this be selected? How should it be set up and tested and what training should be provided?
3. Monitoring—this includes both clinical and ventilator monitoring

### 8.3.2 *Stages in the Management of Transport Ventilation*

Transport ventilation has been the subject of a number of studies which have analysed the various stages in the procedure and the steps and checks that should take place in each. Box 8.2 summarises the stages involved in transport ventilation. These are discussed in more detail below.

#### **Box 8.2 An Overview of the Stages of Transport Ventilation (from Blakeman and Branson 2013a)**

- 1 Training in the use of the ventilator and ancillary equipment

## 2 Pre-use checking of the ventilator and all airway and ancillary equipment

Make sure that the default settings are correct before proceeding with the clinical mission

3. Following an emergency intervention before loading the ventilator dependent patient into the transport vehicle, check the stabilisation of the patient from the emergency response. In many situations a smaller, resuscitation ventilator or a bag–valve device will be used for the emergency response outside the ambulance and the patient will be changed to a more complex ventilator for transport once inside the ambulance

## 4. Plan the changeover to transport

Are sufficient personnel available to move and monitor the patient?

Is a backup ventilation device available in the ambulance if the transport ventilator malfunctions?

## 5. Loading the Patient

Pre- and post loading checks of ventilation.

Connecting the transport ventilator

Obligatory checks of airway, ventilation and patient condition after every movement of the patient.

## 6. Continuing Conduct of Ventilation during Transport

NB some emergency medical services (e.g. SAMU in France ) require the ambulance to stop if problems arise during transport (both with ventilation and other clinical emergencies eg cardiac arrest)

### 8.3.3 *The Logistics of Transport Ventilation*

Transport ventilators may be pneumatically or electrically powered or both. It is therefore essential to ensure that sufficient battery power and backup is available and that compressed oxygen supplies are adequate for the planned journey. Sophisticated turbine ventilators, used in specialist transport use air as the driving gas which is supplemented by oxygen. The oxygen requirements will therefore depend on the set concentration of oxygen (usually 40%) and the minute volume delivered to the patient. In the case of the use of a pneumatic ventilator during transport, the oxygen requirement should be worked out by consulting the manufacturer's handbook which should provide the amount of oxygen that is used in powering the ventilator in addition to that being delivered to the patient. In pneumatic ventilators, the use of the airmix system (Chap. 7) considerably prolongs the life of the oxygen cylinder while providing an inspired oxygen concentration that is appropriate for long term ventilation. As an example Table 8.1 gives endurance times of a Pnuepac Parapac Plus ventilator set to deliver a minute volume of 10 litres per minute with 100% oxygen and 50% from the airmix system.

**Table 8.1** A typical endurance table for a pneumatic ventilator operating from an E sized oxygen cylinder (680 litres) (Reproduced with permission from Smiths Medical International, Luton UK)**No Air-Mix (NAM) Endurance (mins.) 100% O<sub>2</sub> (680L)**

		Frequency (bpm)						
		8	10	12	15	20	25	40
Tidal volume (mL)	100	No PEEP				213	170	106
	200				174	131	105	65
	350			138	111	83	66	41
	500		121	101	81	61	49	
	700	112	89	75	60	45		
	1000	80	64	53	43			
	1500	54	44	36				

**Air-Mix (AM) Endurance (mins.) 50% O<sub>2</sub> (680L)**

		Frequency (bpm)						
		8	10	12	15	20	25	40
Tidal volume (mL)	100	No PEEP				373	298	186
	200				370	278	222	139
	350			335	268	201	161	100
	500		314	262	210	157	126	
	700	305	244	203	163	122		
	1000	228	183	152	122			
	1500	161	129	107				

It will be seen that for a tidal volume of 500 ml at a frequency of 12/min the endurance time increases from 101 min at 100% oxygen to 262 min on air mix.

### 8.3.4 Monitoring of Artificial Ventilation During Transport

Chapter 7 described the monitors available on portable mechanical ventilators. These play an important role during transportation where the circumstances and moving the patient may cause distraction from clinical observation. However, ventilator monitoring systems should be used as an aid to careful clinical observation which is essential at all times during transport.

#### 8.3.4.1 Clinical Observation

Monitoring of the respiratory condition of the patient during transport must be part of careful clinical monitoring of the whole condition of the patient. This will include the cardiac and circulatory status aided by ECG and automatic blood pressure monitoring. Here we consider only monitoring of the respiratory system.

No patient on a ventilator should ever be left unattended in the non – ICU setting. The member of the emergency transport team designated for monitoring artificial



ventilation must follow a systematic pattern of checks at regular intervals. Particular attention is required as to the patency of the airway and circuit whenever the patient is moved (e.g. in and out of an ambulance) or when there is an incident involving the vehicle in which the patient is being transported (e.g. bumpy road in rural areas, sharp deceleration).

The basic clinical monitoring of artificial ventilation involves the clinical skills of inspection and auscultation. These are listed in Box 8.3. Using the stethoscope for the latter which is an essential part of normal clinical practice may be difficult in a moving vehicle and particularly in an aircraft due to the ambient noise.

### **Box 8.3 Clinical Monitoring of the Respiratory System During Patient Transport**

#### **Inspection**

Are there regular chest movements in synchrony with the ventilator?

Is the patient's colour satisfactory?

Is there evidence of cyanosis of mucous membranes?

Has the patient's conscious level changed from that established during the primary and secondary emergency assessments and before loading into the transport vehicle?

Is the airway and patient circuit fixation satisfactory?

Is the circuit and patient valve clearly visible and not covered by other equipment that may cause a blockage?

Is the pilot balloon on the ETT correctly inflated?

Are secretions properly cleared from the oropharynx around the ETT?

If there is a chest drain inserted is it functioning correctly?

#### **Auscultation**

#### **1 Using Ordinary Hearing:**

Is there any air leak audible around the ETT?

Are any ventilator alarms sounding?

#### **2 Using the stethoscope**

Is air entry to the lungs equal and satisfactory on the right and left sides of the chest?

Are there sounds of secretions inside the ETT and the upper main airways?

Note that the instrumental monitoring on the ventilator and ancillary devices must always be used in conjunction with careful clinical monitoring.

#### **General monitoring of the patient during transport.**

General monitoring, such as ECG, oxygen saturation, pulse rate and blood pressure must be integrated with the specific ventilation monitoring listed above.

### 8.3.4.2 Specific Ventilator Monitoring

This is provided by monitors and alarms on the ventilator itself and by the use of ancillary equipment which was discussed in the previous chapter.

The monitoring available on the ventilator being used for transport will depend on the type of device used. The user of the ventilator must be completely familiar with the device and have received careful training in non-clinical settings, for example using a simulator. Until about 30 years ago transport ventilators had very limited instrumental monitoring compared with hospital ventilators. This situation has now changed with the development of complex electronic transport ventilators. However these will only be used by specialist transport teams. Pneumatic ventilators, which are likely to be used by non-specialists also now must be equipped with essential ventilator performance monitoring to qualify for use as a transport ventilator. This is listed in Box 8.4.

#### **Box 8.4 Essential Ventilator Monitoring During Transport Ventilation and Checks to Perform**

##### **Oxygen supply gauge**

This is a pneumatic fish eye device on pneumatic ventilators and should be independent of any electronic monitoring (Chap. 7).

Is the gauge showing white, indicating sufficient driving gas pressure?

Check the ventilator oxygen setting against the pressure in the driving cylinder and regulator.

Battery—life indicator (for electrically driven and electronically—controlled turbine ventilators). Is there sufficient power remaining for the anticipated duration of the transport?

High inspiratory pressure relief valve

This is usually set to 40 or 60 cm H<sub>2</sub>O (Chap. 7) but can be operator adjusted on some ventilators. Check the setting.

Manometer

Is the peak patient inspiratory pressure (PIP) correct (between 15 and 20 cm H<sub>2</sub>O for an average adult)?

Is the PIP too high or too low (>20 cm <10 cm) accompanied by sounding alarms?

Is the rise pattern of the PIP appropriate for volume or pressure targeted ventilation (Chap. 7). (More sophisticated ventilators have a pressure loop display which gives information about the pressure and delivered volume of delivered breaths).

Expired tidal volume

Many transport ventilators have a tidal volume monitor which monitors expired tidal volume.

Is the expired tidal volume equal to that of the set inspired tidal volume (indicating a possible leak in the circuit)?

Is the expired tidal volume appropriate for the size of the patient being ventilated? (10 ml/kg) but  $V_t$  values are falling for emergency and transport ventilation in line with current ICU practice and ILCOR resuscitation guidelines which state that the  $V_t$  should be between 500 and 600 ml for a normal adult.

Does the frequency setting of the ventilator produce a satisfactory minute volume with the expired  $V_t$ ?

Alarms

Are any alarms sounding or flashing?

Has the alarm mute button (if fitted) been activated?

Is the ventilator cycling indicator showing correctly?

Is the patient demand light (operates when the patient takes a spontaneous breath while being artificially ventilated) showing?

Capnography

Is the expired  $\text{CO}_2$  waveform satisfactory? (see Chap. 5)

Is the level of expired  $\text{CO}_2$  satisfactory? (normally between 30 and 36 mm Hg (4 and 4.8 kPa))

Pulse oximetry

Is the saturation  $>93\%$  on the set  $\text{FiO}_2$ ? (Use the minimum possible inspired oxygen concentration to obtain this level of saturation).

Is the pulse rate too high or too low?

Ventilator adaptation to the patient's own efforts (SIMV and SMMV).

If the patient is making spontaneous breathing efforts in partial respiratory failure is the ventilator adaptive system (e.g. SIMV) responding correctly or is the patient tending to breath against the ventilator ('fighting' the ventilator)?

## 8.4 Potential Problems During Transportation of Patients Who Are Being Ventilated

Several studies have examined the incidence of adverse effects during transport ventilation (see suggestions for further reading). Adverse effects may be defined as events occurring during transport which are potentially detrimental to the stability of the patient and which often require medical intervention.

Figures for the incidence of adverse effects for intrahospital transport ventilation range from between 5.9 to 84%. However, this range of incidence covers a large time span and a number of different circumstances. With the development of transport ventilation protocols in recent times it is likely that the incidence of adverse effects is diminishing.

### **8.4.1 *Causes of Adverse Effects***

Adverse effects during transport ventilation can be related to:

#### **1 Equipment Failure**

This includes:

Patient circuit disconnection

Displaced or occluded endotracheal tube

Accidental extubation during the movement of the patient

Failure of battery power in ventilators that are power – dependent.

Failure of the compressed oxygen supply

Accidental alteration of the ventilator control settings

#### **2 Patient–Related Events**

Worsening of the underlying pathology requiring artificial ventilation

Pneumothorax (possibly as a result of hyperventilation of the patient)

Insufficient sedation of a partially conscious patient

Biting on the endotracheal tube (a Guedel airway should be inserted alongside the tube to prevent this)

Occlusion of the airway by secretions with inappropriate suction

Inappropriate synchronisation of the partially–breathing patient with delivered ventilator breaths

Inadequate clinical surveillance of a patient on a ventilator

## **8.5 Conclusions**

- 1 Transport ventilation is different from emergency ventilation and concerns the safe movement of a patient who is dependent on a ventilator but in a stable condition, from one location to another.
- 2 There are inherent risks in transport ventilation associated with both the location and the movement of the patient.
- 3 Transport ventilation over long distances is usually controlled by specialists using relatively sophisticated ventilators. However it is important that non-specialists providing basic transport ventilation should have an understanding of specialist transport ventilation.
- 4 Guidelines for the safe conduct of transport ventilation have been issued and these should be supplemented by special training for those involved.
- 5 Before starting the transport equipment availability and function should be verified according to standard checklists. The logistics of provision of oxygen during the transport should be calculated.

- 6 Careful monitoring of the patient is essential during transport, no matter what is the distance involved. This should be based upon clinical skills of observation and auscultation, supplemented by the information given from ventilator monitoring systems and alarms.
- 7 A supplementary means of ventilation should be available at all times and used when any unexplained problem arises in mechanical ventilation.
- 8 Problems arising during transport ventilation can be related to the equipment used and changes in the patient's condition. With better adherence to standard practice protocols the incidence is thought to be falling but this trend can only be continued by constant awareness of the potential dangers of transport ventilation and continued training.

## Suggestions for Further Reading

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# Chapter 9

## Paediatric Artificial Ventilation for the Non-Specialist



### 9.1 Introduction

Neonatal and infant ventilation in the hospital is the realm of the paediatric intensive care physicians and nurses in special care baby units and other facilities. However in emergency situations, in developing nations and remote settings where medical care is provided, including disaster medicine, non-specialist medical personnel may have to provide paediatric artificial ventilation and other respiratory support. Thus, an understanding of the essential differences between ventilating adults and neonates, infants and children is important within the scope of this book. This chapter discusses the basic differences between adult and infant respiratory physiology with an overview of the essential respiratory changes that take place at birth. The important area of paediatric resuscitation is covered with a synopsis of the current ILCOR guidelines in this area.

The recognition and management of airway obstruction, both upper and lower is also considered with details of the technical options available for paediatric ventilation. The complications of neonatal and infant ventilation are considered to give an insight into the work of the specialist neonatal units and hospital ventilator care. Finally, we discuss the problems associated with paediatric transport ventilation and the options available for mechanical ventilation and the suitable modes required.

### 9.2 Definitions

The overall term ‘paediatric’ comprises the following sub sections:

- Neonate—the first 28 days of life or less than 44 weeks post conception
- Infant—1 month to 1 year
- Child—greater than 1 year to adolescence.

### 9.3 Overview of the Differences Between Adult and Paediatric Respiratory Anatomy and Physiology

There are considerable differences between the paediatric and adult respiratory systems. These are particularly marked for neonates. In practical terms these affect both mask ventilation and ventilation via an endotracheal tube.

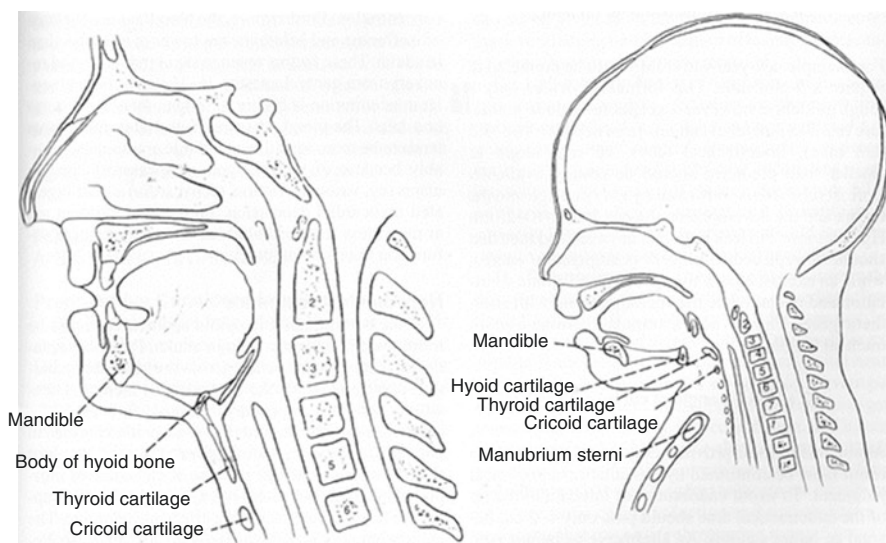
#### 9.3.1 Anatomical Differences

The differences between the anatomy of the airway in adults and infants is shown in Fig. 9.1 and may be summarised as follows:

In the neonate and infants:

1. There is a proportionally larger head
2. The tongue is proportionally larger
3. There are narrow nasal passages
4. The structure of the larynx is different (at a level of C4 rather than C6 in adults) with a longer epiglottis

All these factors make neonates and infants obligatory nasal rather than mouth breathers. An important anatomical point which affects intubation is that the narrowest part of the airway in children younger than 5 years is the cricoid cartilage (as opposed to the space between the vocal chords, the rima glottidis, in adults). The



**Fig. 9.1** Anatomical differences in the airways of adults and infants

narrow trachea in children makes them very vulnerable to large airway blockage by oedema of the walls. Even 1 mm of oedema has a proportionally greater effect on the airway resistance in children compared with adults. This is particularly important in allergic reactions.

Other anatomical differences which decrease the efficiency of respiration in neonates and infants are relatively weak intercostal and diaphragmatic muscles which are underdeveloped compared with adults, pliable ribs and a protuberant abdomen.

The implications of these anatomical differences for airway management in neonates infants and children are summarised in Box 9.1.

### **Box 9.1 Implications of Anatomical Differences Between Adults and Infants for Airway Management**

The shape of the face and mouth requires specially contoured masks which minimise dead space.

The prominent occiput tends to cause neck flexion which can be overcome by placing a rolled towel under the shoulders.

The oversized tongue requires the use of oral airways (a miniature Guedel airway). A nasal airway can traumatize small nares or prominent adenoids.

Because of the length of the epiglottis, a straight blade laryngoscope is usually used in hospital practice.

The required diameter and length of an endotracheal tube in children is calculated using the following formula:

$$\text{Tube diameter (mm)} = 4 + \text{age}/4$$

Note that this formula does not apply to premature and full-term neonates where a 2.5–3 mm and 3–3.5 tube is required respectively.

$$\text{Cut endotracheal tube length (cm)} = 12 + \text{age} / 2$$

## **9.3.2 Physiological Differences**

### **9.3.2.1 Age-Related Differences in Physical Vital Signs**

There are age-related changes in physiological vital signs which are presented in Table 9.1. A basic knowledge of these is important in paediatric clinical assessment.

### **9.3.2.2 Cardio-Respiratory Physiological Changes at Birth**

There are important physiological changes at birth between the foetal and neonatal cardio-respiratory system. Although this text does not primarily concern the cardiovascular system, in the context of the neonate both this and the respiratory systems



**Table 9.1** Age-related changes in physiological vital signs

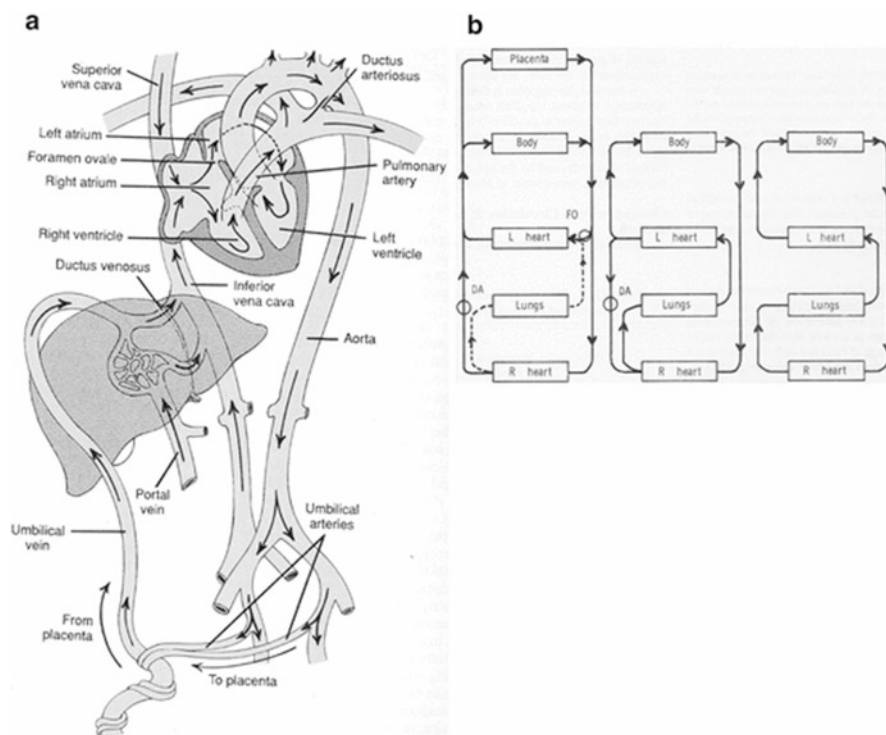
Age	Respiratory Rate (/min)	Heart rate (/min)	Systolic blood pressure	Diastolic blood pressure
Neonate	40	140	65	40
12 months	30	120	95	65
3 years	25	100	100	70
12 years	20	80	110	60

must be considered together. In the uterus, the placenta is responsible for oxygenation of the foetal blood from the oxygen carried in the maternal circulation. Most of the blood flow arriving at the heart from the inferior vena cava (note—in the foetus this is oxygenated blood) is directed into the left ventricle via a window called the foramen ovale. From the left ventricle this oxygenated blood passes into the aorta and then to the rest of the body. Blood arriving from the superior vena cava (which is deoxygenated) goes into the pulmonary artery (PA) and the ductus arteriosus (a vessel connecting the PA to the aorta. The blood flow through the lungs in the foetus is minimal. At birth this situation quickly changes and the foetal circulation becomes adult. The following events occur at birth (1) the placenta is removed from the circulation (2) the portal blood pressure falls and the ductus venosus closes. Blood then becomes oxygenated in the lungs (3) when the ductus arteriosus is exposed to oxygenated blood it closes (4) the lungs expand and the pulmonary resistance rapidly decreases while peripheral resistance rises (5) the increase in pressure in the left side of the heart caused by the increase in peripheral resistance causes the foramen ovale to close. Sometimes this closure is not complete, giving rise to a ‘hole in the heart’ baby.

This changeover of circulation occurs at a critical period and following birth the infant can easily flip from adult back to a foetal circulation. This situation is called transitional circulation and can be induced by many factors including hypoxia and hypercapnia. Figure 9.2 shows the foetal circulation diagrammatically.

### 9.3.2.3 Lungs

Infants rely mainly on diaphragmatic breathing and their muscles are more likely to tire than in adults. The compliance of the chest wall is higher than in adult, which means there will be major visible in—drawing of the ribs in the presence of any airway obstruction. The compliance of the lung tissue however is lower than that of the adult. Surfactant plays a major role in the reduction of surface tension inside the infant alveoli and keeping the lungs open. If there is an absence of surfactant, as in the case of premature babies the lungs cannot expand properly and the neonate must be ventilated inside a special care baby unit until the surfactant builds up. This condition is called the neonatal respiratory distress syndrome. Note that this syndrome



**Fig. 9.2** (a) the foetal circulation in utero. (b) Diagrammatic representation of the circulation in the foetus, the newborn and the adult (DA ductus arteriosus, FO foramen ovale)

is essentially caused by a lack of surfactant unlike the adult respiratory distress syndrome which has many causes. Both conditions however cause hypoxia through a type 1 respiratory failure. Because of the low lung compliance in the infant, a negative pressure of 40–80 cm H<sub>2</sub>O is required for lung expansion compared with the few cm H<sub>2</sub>O of negative pressure required in normal adult breathing.

#### 9.3.2.4 Tidal Volume

The tidal volume of the infant gradually increases with age but if adjusted for body weight is relatively constant at 5–7 ml/kg. To ensure an adequate minute volume in an infant with a small tidal volume, the respiratory rate is therefore higher. In hospital neonatal ventilation higher tidal volumes (between 10 and 15 ml/kg) are usually set, particularly if an uncuffed ETT is being used, where there is often a leak during the inspiratory phase.

### 9.3.2.5 Anatomical Dead Space

The anatomical dead space in an infant is calculated as  $0.3 \times V_t$ . As in the adult this is the proportion of the tidal volume with each breath which does not take part in oxygen exchange in the lungs.

### 9.3.2.6 Functional Residual Capacity

The functional residual capacity in an infant is much lower than in an adult. There is therefore less buffering capacity and rapid desaturation of the blood can occur with the onset of respiratory failure.

## 9.4 Respiratory Failure and Cardiac Arrest in Neonates and Infants

Cardiac arrest occurs because the heart is starved of oxygen. In adults the most common cause of cardiac arrest is the occlusion of the coronary arteries by bleeding into a plaque of fatty tissue that has built up into the wall of the artery (coronary thrombosis). In infants and children the heart also stops when deprived of oxygen but in this case the hypoxia is due to respiratory arrest which precedes cardiac arrest. In trauma it is also a consequence of circulatory insufficiency following blood loss. Since respiratory deficiency and arrest is treatable with artificial ventilation it is therefore vital to be able to identify the signs of respiratory arrest in infants and children and take immediate remedial action. This is particularly important since the outcome of cardiac arrest in children outside the hospital is even worse than in adults. This is due not only to the effects of hypoxia and acidosis on the heart but also to hypoxic damage to the cells of key organs such as the brain and kidneys.

### 9.4.1 *Causes of Respiratory Failure in Infants and Children*

Chapter 4 considered the causes of respiratory failure in adults. Respiratory failure in neonates, infants and children forms a special subclass. The causes of respiratory failure in infants and children may be classified as:

1. Traumatic—with effects on the respiratory centre in the brain and damage to the chest wall and airways
2. Infective—as a result of overwhelming lung infection or meningitis
3. Drug induced—opioids in an incorrect dose will cause depression of the respiratory centre in the medulla of the brain
4. Toxic—a number of toxic chemicals can induce respiratory arrest

5. In addition, there may be physiological causes. The respiratory system of premature babies and neonates may not be sufficiently developed to ensure normal breathing after birth.

### **9.4.2 Recognition of Paediatric Respiratory Failure: Primary and Secondary Survey**

#### **9.4.2.1 Airway**

Early recognition of respiratory failure and impending arrest is vital. As with adults this must be done immediately as part of the primary patient survey in emergency using a ‘look, listen and feel’ approach. The first thing is to ensure the patency of the airway. If the infant or child is able to speak or cry this is an indication that the airway is open. In the case of management of mass respiratory arrest (a possible consequence of a chemical agent attack) a good rule is to attend to the patients who are silent first. The normal means of supporting the airway should be applied, but remember not to overextend the neck as this in itself can cause airway obstruction in the child.

Before using a stethoscope, listen at the mouth for added sounds such as inspiratory stridor which indicates upper airway obstruction or wheezing, which indicates lower airway obstruction. If there are gurgling sounds, these indicate the need for suction to clear secretions. Snoring sounds indicate a partially obstructed upper airway due to inadequate positioning.

#### **9.4.2.2 Breathing**

The adequacy of breathing efforts can be rapidly assessed by observation. The normal respiratory rate in neonates and infants changes with age as shown in Table 9.2. The respiratory rate should therefore be counted taking into account the patient’s age. A falling respiratory rate in the presence of other signs which indicate increased work of breathing (in—drawing of the chest wall, flared nostrils, use of accessory muscles and expiratory grunting) indicates imminent respiratory arrest. If the degree of respiratory failure is less the air entry to both lungs can be assessed using the stethoscope.

**Table 9.2** Normal respiratory rates in infants and children

Age (years)	Breaths per minute
Less than 2	30–40
2–5	25–30
5–12	20–25
Over 12	15–20

### 9.4.2.3 Other Systemic Indicators of Respiratory Insufficiency

In addition to observational and auditory indicators of the respiratory system itself there are a number of other systemic indicators which must be considered. These are:

1. Skin and mucous membranes—cyanosis indicates very severe hypoxia in infants and children
2. Heart rate—initially hypoxia causes a tachycardia until the heart muscle and conducting systems are so badly affected that they begin to fail and the heart slows. Bradycardia (heart rate lowering) in this situation indicates impending cardiac arrest.
3. Mental status—mild degrees of hypoxia can cause drowsiness or changes in the behaviour of the child.

## 9.5 Management of Airway and Ventilation in Paediatric Respiratory Arrest

### 9.5.1 Upper Airway Obstruction

When there are signs of respiratory failure and arrest as indicated above immediate steps should be taken to clear and support the upper airway and to start artificial ventilation. An attempt to open the airway by positioning the head is the first step. As in the adult, head extension helps to open the oropharynx but care should be taken not to overextend. In an infant the head should be in a neutral position. In the child it should be in a sniffing position. If there is trauma only jaw thrust should be used. If there is an indication there might be a foreign body in the oropharynx the finger sweep technique should not be used since it might push the foreign body further down or cause trauma to the soft palate.

The importance of effective suction cannot be overemphasised. The suction apparatus should be mechanical and not compressed gas operated (oxygen is often used in this situation, leading to a waste of a precious gas). A pistol grip sucker such as the Vitellograph (Chap. 6) with a suitable small suction tube gives the best control over suction. If an infant Yankauer sucker is being used there is a small hole in the side to control the vacuum pressure.

Once the airway has been cleared of secretions the next step is to control the tongue (which is relatively large in the case of neonates and infants) using an oropharyngeal (Guedel) airway (Chap. 5) These are available in a number of sizes. The correct size is that which reaches from the centre of the incisor teeth to the angle of the jaw when the airway is placed against the face with concave side up. The airway is inserted in the same way as in the adult with the concave side initially upwards and then rotated to lie adjacent to the tongue. In the case

of young infants the tongue should first be depressed with a tongue depressor or a laryngoscope blade.

### **9.5.2 *Securing the Airway***

Once the airway has been cleared and controlled using the methods described previously it must be secured before effective and controlled artificial ventilation starts. How this is done depends on the training and expertise of the responder. It is important not to waste time with fruitless attempts at intubation when ventilation using a mask is possible and will overcome the life-threatening hypoxia. Generally, in pre-hospital neonatal and paediatric emergencies intubation by non-experts is contraindicated. The technique requires considerable skill and may prolong the time taken until evacuation to hospital is possible.

For responders in difficult circumstances when there are no other medical support options the following points are important about paediatric intubation:

#### **9.5.2.1 Size of the Tube**

The correct dimensions of a paediatric endotracheal tube are:

$$\text{Internal diameter (mm)} = (\text{age} / 4) + 4$$

$$\text{Length at the lips (cm)} = (\text{age} / 2) + 12$$

The technique of intubation is similar to that in adults (Chap. 5) but an uncuffed tube is usually used to prevent oedema at the level of the cricoid cartilage. The tube must be securely taped into position after having confirmed that it is correctly placed by auscultation and observation of chest movements.

## **9.6 Intermittent Positive Pressure Ventilation in Neonates, Infants and Children**

Having secured the airway, if there is only partial respiratory failure free flow oxygen may be used. However, if there is clear respiratory failure or arrest or if there is any doubt about the effectiveness of breathing while breathing oxygen artificial ventilation is essential. In neonates, infants and children, as with adults artificial ventilation is provided using either a self-reforming bag or mechanical ventilators. Expired air ventilation is also possible in emergency and this is considered in the guidelines for the management of paediatric cardiorespiratory failure which are considered in the next section.

### 9.6.1 Bag Valve Mask Ventilation

Bag valve mask ventilation is the technique most often used in emergency paediatric ventilation. However it has potentially the same problems of hypo and hyperventilation which were discussed in the adult context in Chap. 7.

For BVM ventilation in children a suitable mask and bag system must be used (Fig. 9.3). The mask for neonates is round, unlike the adult version. The mask should be applied to the face using a jaw thrust grip to help open the airway with the thumb holding the mask. Ensuring that there is an adequate seal between the mask and face the bag should be squeezed gently at a rate of 15–40 breaths per minute depending on the age (Table 9.2). The chest movements should be carefully observed to avoid hyperventilation and barotrauma from excessive compression of the bag. Free flow oxygen is fed to the bag via a reservoir bag. Effective ventilation will be indicated by the colour of the skin and mucous membranes supported by pulse oximetry readings.

#### 9.6.1.1 Paediatric Life Support

As with adults, artificial ventilation is a key part of cardiopulmonary resuscitation in neonates, infants and children. Ventilation is particularly important since cardiac arrest in these age groups is usually secondary to respiratory failure and hypoxia. As noted above, adult cardiac arrest usually occurs before respiratory arrest as a result of failure of the coronary artery circulation to the heart (coronary thrombosis).

**Fig. 9.3** Paediatric bag-valve-mask device. (Photograph courtesy of Smiths Medical International, Ashford, United Kingdom)



Paediatric cardiopulmonary resuscitation is a special section of the ILCOR international guidelines. There are differences between the adult and paediatric guidelines which are of importance. The references to the 2010 and 2015 guidelines are given in suggestions for further reading at the end of this chapter. Airway and respiratory support must be considered together with cardiac support. The 2015 guidelines for neonatal airway and ventilation support are summarized in Box 9.2.

**Box 9.2 ERC Guidelines for Neonatal and Infant Airway and Ventilation Support (I.K. Maconochie et al./Resuscitation 95 (2015) 223–248)**

If breathing is not normal or absent:

- Carefully remove any obvious airway obstruction.
- Give five initial rescue breaths.
- While performing the rescue breaths note any gag or cough response to your action. These responses or their absence will form part of your assessment of ‘signs of life’,

Rescue breaths for an infant

- Ensure a neutral position of the head as an infant’s head is usually flexed when supine. This may require some extension (a rolled towel/blanket under the upper part of the body may help to maintain the position) and a chin lift
- Take a breath and cover the mouth and nose of the infant with your mouth, making sure you have a good seal. If the nose and mouth cannot be covered in the older infant, the rescuer may attempt to seal only the infant’s nose or mouth with his mouth (if the nose is used, close the lips to prevent air escape)
- Blow steadily into the infant’s mouth and nose for about 1s, sufficient to make the chest visibly rise.
- Maintain head position and chin lift, take your mouth away from the patient and watch for the chest to fall as air comes out.
- Take another breath and repeat this sequence five times.

Rescue breaths for a child over 1 year of age

- Ensure head tilt and chin lift
- Pinch the soft part of the nose closed with the index finger and thumb of your hand on the forehead.
- Allow the mouth to open, but maintain chin lift
- Take a breath and place your lips around the mouth, making sure that you have a good seal.
- Blow steadily into the mouth for about 1s, watching for chest rise
- Maintain head tilt and chin lift, take your mouth away from the patient and watch for the chest to fall as air comes out.



- Take another breath and repeat this sequence five times. Identify effectiveness by seeing that the child's chest has risen and fallen in a similar fashion to the movement produced by a normal breath.
- For both infants and children, if you have difficulty achieving an effective rescue breath the airway may be obstructed:
- Open the child's mouth and remove any visible obstruction. Do not perform a blind finger sweep.
- Reposition the head. Ensure that there is adequate head tilt and chin lift but also that the neck is not over-extended.
- If head tilt and chin lift has not opened the airway, try the jaw thrust method.
- Make up to five attempts to achieve effective breaths., If still unsuccessful, move on to chest compressions.



### 9.6.2 Mechanical Ventilation

Several ventilators have been produced for neonatal, infant and paediatric use and are capable of producing a better quality ventilation than that of the bag-valve mask. The important principle behind paediatric ventilation is that, because of the fragile nature of the lung tissue pressure generation is required rather than volume generation. The differences between these were discussed in Chap. 6. Paediatric ventilators like adult ventilators vary in complexity and some of the more recent electronically controlled turbine ventilators are capable of ventilating both adults and children down to neonates. Most adult pneumatic ventilators are only certified

for use down to a weight of 10 kg. An important point to note about volume - generating pneumatic ventilators is that when operating at the extreme lower range of the capability they become pressure rather than volume generators which makes their use in an emergency when no paediatric ventilator is available possible. It is also possible to use a volume generator with a variable pressure release (APL) valve. This is a technique used by anaesthetic and intensive care specialists in certain circumstances (Fig. 9.4)


Paediatric ventilators, particularly those used in transport ventilation have more complex features and modes than basic adult ventilators. A typical example of a pneumatic paediatric ventilator is the Pneupac babyPac (shown in Figs. 9.5 and 9.6). The babyPac pneumatic ventilator is designed to ventilate neonates and infants and therefore to deliver small tidal volumes safely. To do this, it operates as a time cycled, pressure limited ventilator. In this type of ventilation the controlling

**Fig. 9.4** Infant anaesthesia in a remote location using a modified Pneupac CompPac autonomous ventilator. This volume—preset flow generator has been converted to a neonatal pressure generator by inserting a spill (APL) valve into the patient circuit. (Photograph courtesy of Smiths Medical International, Luton, United Kingdom and Dr. Graham Bell)




**Fig. 9.5** The Pneupac babyPac neonatal and infant ventilator. (Photograph courtesy of Smiths Medical International, Luton, United Kingdom)





Before use read User's Manual  
**WARNING**

This equipment should only be used by trained personnel




### babyPAC™ 100 VENTILATOR

*Time cycled pressure generator ventilator for neonates and infants - with pressure monitoring*

#### NEONATE / INFANT


**Not to be used on unattended patients**  
**Not for use in Critical Care**


**Basic Instructions**

1. Connect to gas supply and select function- CMV, IMV or CPAP.
- Check pressure indicators:
-  - indicates low supply- O<sub>2</sub> or Air (will operate on one supply gas only)
2. Set  $V_{TDEL}$  by means of  $T_{INS}$  &  $\hat{P}$
3. Set Freq or I:E by means of  $T_{EXP}$  &  $T_{INS}$
4. Set Alarm below  $\hat{P}$  & test by occluding patient connection. Test low press/disconnect alarm by opening patient connection
5. Set Alarm to safe level, but above  $\hat{P}$
6. Set O<sub>2</sub> concentration as required

#### Controls:

$T_{INS}$  Inspiratory time (sec)  
 $T_{EXP}$  Expiratory time (sec)  
 $P$  Inspiratory press (cmH<sub>2</sub>O)  
 $CMV$  Controlled ventilation  
 $PEEP$  Positive end expiratory pressure (cmH<sub>2</sub>O)  
 $CPAP$  Continuous positive airway pressure (cmH<sub>2</sub>O)  
 $IMV$   $T_{EXP}$  expanded x10  
 $Alarm$  High press alarm setting with press relief (cmH<sub>2</sub>O)

 Settings requiring caution and which give highest usage of compressed gas

 For complete instructions refer to user's manual.


If problem occurs refer to User's Manual or contact your local service representative. **DO NOT ATTEMPT to service the unit yourself.**

US Tel: 1-800-558-2345

**Frequency (1/min)**

	0.25	0.35	0.50	0.70	1.00	2.00
$T_{EXP}$	120	100	80	63	48	27
0.50	80	70	60	50	40	24
1.00	48	44	40	35	30	20
2.00	27	26	24	22	20	15
4.00	14	14	13	13	12	10
$T_{INS}$	0.25	0.35	0.50	0.70	1.00	2.00


Protected by Patents



Testing has confirmed compatibility at 3 Tesla and 430G/cm with RF at 0.82W/kg and 125.5MHz using a Shielded magnet, but intended use is with this control module away from magnet bore

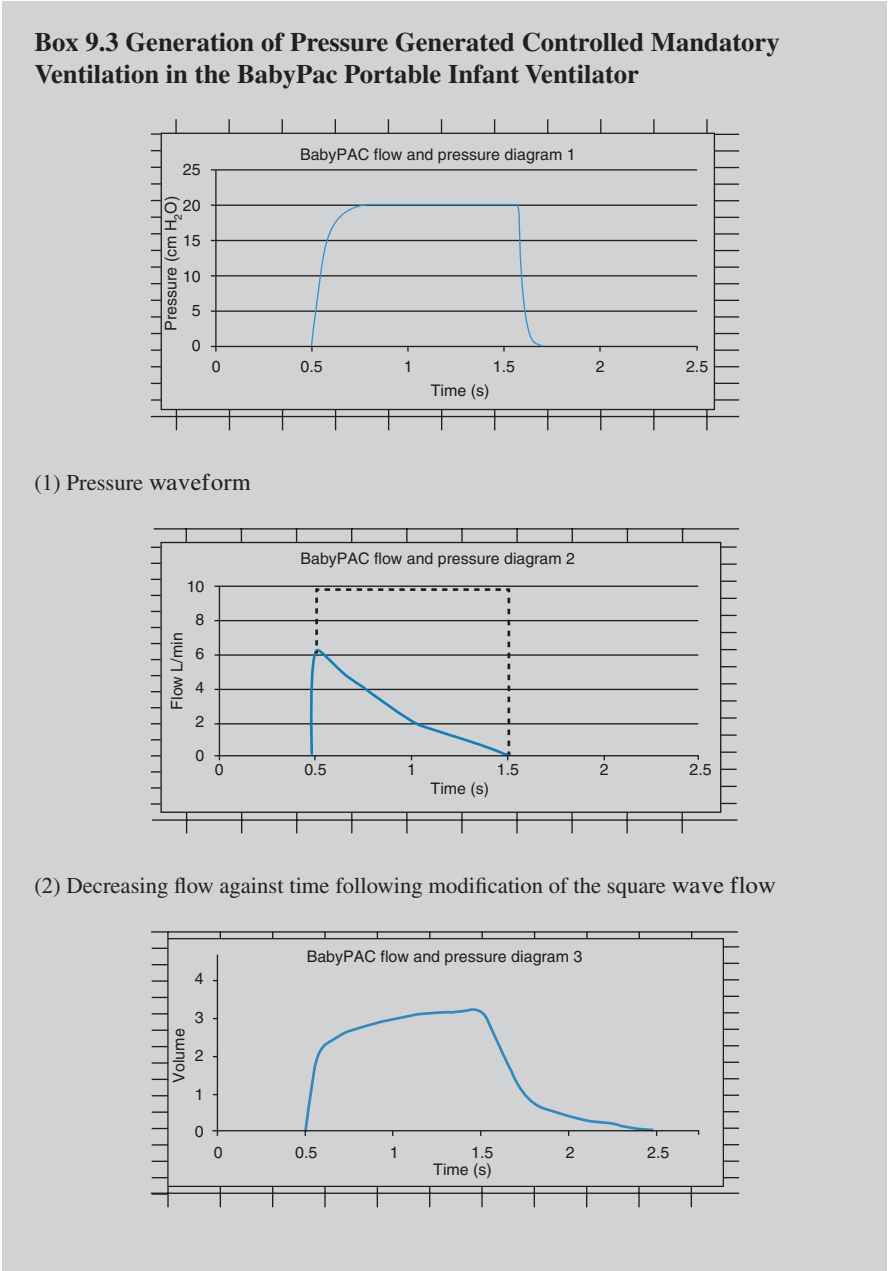
## smiths medical

Smiths Medical PM, Inc., N7 W22025 Johnson Drive  
Waukeasha, WI 53186-1856  
UK Tel: (+44) (0)1582 430000

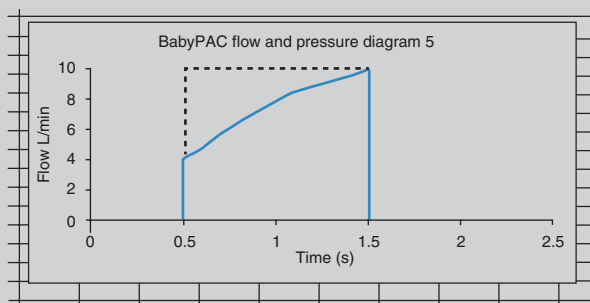

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**Fig. 9.6** Table of settings for inspiratory and expiratory times to produce a given frequency of ventilation. Since the ventilator is a pressure generator the delivered tidal volume will be a function of the compliance and resistance of the lungs and airway. Inspiratory and expiratory times are known as independent variables of ventilation (ie they are set by the operator). The pressure setting for ventilation is also an independent variable. (Photograph courtesy of Smiths Medical International, Luton, United Kingdom)

parameter is the airway pressure during inspiration. In computer - controlled ICU ventilators the flow rate is varied during inspiration to generate the required inspiratory pressure. The inspiration terminates at the end of a set inspiratory time. Since the inspiratory flow rate varies, the tidal volume delivered at the end of the inspiratory phase depends on the compliance of the lungs and the system delivering the ventilation. The babyPac is essentially a pressure generator. Details of how this pressure generation is produced are shown in Box 9.3.



(3) Delivered tidal volume against time during inspiration.



(5) Diverted flow during inspiration

Babypac is a pneumatic ventilator which is time cycled using a patent pneumatic oscillator. During inspiration the ventilator generates a square waveform (diagram 1) for flow at 10 l/min which is delivered to the inspiratory limb of the patient circuit. This square flow wave is turned into the decreasing flow waveform (diagram 2) by using the Y piece configuration of the babyPac circuit. As the pressure builds up in the patient's airway an increasing proportion of the square wave—generated flow is released into the expiratory limb of the circuit and back to the ventilator until there is no flow at all. The curve (diagram 3) shows the proportion of the flow that is diverted (from 4 l/min at the beginning of inspiration to 10 l/min at the end. This ensures the decreasing flow pattern (and the required pressure limitation during inspiration. The resulting characteristic pressure waveform is shown in diagram 4 and the resulting delivered tidal volume in diagram 5.

It should be noted because of the special way babyPac generates its flow waveform it should only be used its special Y piece circuit and never with the single limb patient circuit and valve used on other Pneupac ventilators which operate as time cycled, volume preset flow generators designed primarily for adult ventilation (Fig. 9.7).



**Fig. 9.7** The Y shaped circuit of the Babypac connected to an LMA, ETT and infant pharyngeal mask. The inspiratory and expiratory arms of the Y circuit carry fresh gas from the ventilator and take back expired air which is discharged from the ventilator to the atmosphere. (Photographs courtesy of Smiths Medical International, Luton, United Kingdom)

### 9.6.2.1 Non-Pneumatic Paediatric Ventilators

The ventilator described in the previous section is portable and designed for emergency and transport use. In recent years there has been considerable development of small electronically controlled ventilators which have many of the features found on a complex ICU ventilator. The Dräger Babylog VN500 shown in Fig. 9.8 is an example. These allow an adaptive ventilation which supports the neonate and infant respiratory effort. Such ventilators depend on a turbine to provide the positive inflation pressure and computer control of the timing and adaptation of the ventilation. This type of ventilator is relatively complicated to set up and should only be used by medical and nursing personnel and respiratory therapists who have specialised knowledge of their function. It is not recommended for non-specialists.

## 9.7 Paediatric Continuous Positive Airway Pressure

Continuous positive airway pressure as a support for breathing that is still present but not sufficient to provide adequate ventilation of the lungs was discussed in Chap. 6. CPAP has an important role to play in paediatric respiratory support. Remember that CPAP is not a ventilation mode and if breathing stops it will not provide artificial ventilation. In paediatric ventilation, CPAP is usually accompanied by an apnoea alarm placed beneath the patient which will alert the operator to

**Fig. 9.8** The Dräger Babylog VN 500 ventilator.  
(Photograph © Dräger France SAS, France)



respiratory arrest. As always, in the transport setting such devices do not replace the requirement for careful clinical observation.

### ***9.7.1 Delivered Oxygen Concentration in Paediatric Ventilation***

As with adult oxygen therapy and IPPV the oxygen concentration delivered to the patient should be reduced to the minimum compatible with maintaining good arterial oxygenation. This is usually between 40 and 50%. Control of delivered oxygen concentration is particularly important in neonates where free radical generation in the lungs can worsen RDS. In addition, high concentrations of oxygen can cause damage to the eyes, a condition known as retrolental fibroplasia. In transport ventilation of neonates and infants measuring the oxygen saturation of the blood using a pulse oximeter is a practical way of monitoring which is within the scope of non-specialist care.

## **9.8 Conclusions**

1. Artificial ventilation of neonates, infants and children in emergency and transport is usually controlled by specialists.
2. However, there are situations such as the need for resuscitation, mass artificial ventilation during disasters and epidemics and working in remote locations, where the non-specialist may be required to provide ventilator care. Thus, a basic knowledge of paediatric ventilation is important.
3. There are a number of important differences in anatomy and physiology between adults, neonates and infants which are relevant to the provision of artificial ventilation
4. Respiratory failure in children is the main cause of cardiac arrest. It has a number of causes including traumatic, infective and toxic. Recognition of primary respiratory failure using both clinical and technical means is important.
5. Management of acute respiratory failure in children involves both airway and ventilation skills as with adults. However, the equipment used is smaller and specialised.
6. Basic paediatric life support is controlled by the ILCOR resuscitation guidelines. Rescue and bag-valve ventilation both have an important role. The problems associated with bag-valve ventilation are essentially the same as those in the adult.
7. Mechanical ventilation of neonates, infants and children is provided by a range of usually complex electronically-controlled ventilators operated by intensive care and anaesthetic specialists, together with respiratory therapists in the USA. In addition there are a number of pneumatic devices which deliver a more limited range of respiratory support. Pressure-controlled ventilation is used to prevent damage to delicate neonatal and infant lungs.



8. Control of oxygen delivery in paediatric practice is as important as in adults. In addition to possible neural and lung damage from free radicals there is also the well-recognised problem of retrolental fibroplasia, caused by high  $PO_2$  values with prolonged ventilation of neonates at high oxygen levels. This can be a cause of blindness. Inspired oxygen should be titrated to give an arterial saturation of between 94 and 98%.
9. In emergency ventilation, the treatment of hypoxia should take priority over concerns about oxygen toxicity.

## Suggestions for Further Reading

- Atkins DL, Berger S, Duff JP, et al. Part 11: pediatric basic life support and cardiopulmonary resuscitation quality: 2015 American Heart Association guidelines update for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation*. 2015;132(suppl 2):S519–25.
- de Caen AR, Berg MD, Chameides L, et al. Part 12: pediatric advanced life support: 2015 American Heart Association guidelines update for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation*. 2015;132(suppl 2):S526–42.
- Maconochie IK, Bingham R, Eich C, et al. European resuscitation council guidelines for resuscitation 2015 section 6. Paediatric life support Resuscitation. 2015;95:223–48.
- The 2019 UK Ambulance Services Clinical Practice Guidelines (JRCALC Clinical Practice Guidelines). <https://www.aace.org.uk>. Accessed 20 May 2020.



# Chapter 10

## Artificial Ventilation in Difficult and Extreme Environments



### 10.1 Introduction

Artificial ventilation is usually provided in circumstances with which the operator is familiar. This is the case in the operating theatre or intensive care unit of a hospital, in an ambulance or in a hospital emergency room. Equipment, training and backup are usually standardised and the provision of ventilation care is not affected by changes in the surrounding environment.

However, emergency medical care, including artificial ventilation is often required in situations where the surrounding environment is very much altered from the familiar circumstances of everyday life in developed countries. This situation may be termed ventilation in difficult and extreme environments.

Ventilation in extreme and difficult environments includes the following situations:

- (1) following natural and man-made disasters, either in developed countries where the normal medical infrastructure may be severely disrupted by, for example an earthquake or in developing nations where the medical response system is rapidly overwhelmed due to its having been essentially basic before the disaster.
- (2) during respiratory epidemics such as SARS and COVID19
- (3) in toxic environments following accidental or deliberate release of toxic industrial chemicals or following exposure to products of combustion including smoke and carbon monoxide
- (4) in hyper- and hypobaric environments: these include pressurised cabins of aircraft, decompression chambers and high mountains
- (5) in everyday medical practice in developing nations and in remote parts of the world where supplies and support may be strictly limited

Provision of artificial ventilation in any of these cases may be for an individual or multiple patients. This will involve the need for mass ventilation which is the

subject of Chap. 12. In all the situations listed above, specialist ventilatory care provide by intensive care doctors and nurses and also by respiratory therapists (in the United States) may not be available. Non-specialist medical and nursing personnel should therefore be familiar with the essentials of what is possible in these difficult situations and be trained and prepared to respond when they arise.

This chapter addresses the situations listed above which affect civilian medical practice. The organization of military battlefield ventilation, although having many common problems with civilian operations is a specialized situation which is outside the scope of this book and interested readers are referred to specialized texts.

## **10.2 Artificial Ventilation Following a Disaster**

Natural disasters occur in all parts of the world. In many developing nations, disasters such as earthquakes are a frequent event and have serious consequences for an often fragile infrastructure. But natural and man-made disasters also occur in developed nations where a relatively sophisticated medical infrastructure can be reduced to a basic level by effects upon essential services such as the power and compressed gas supplies.

Natural disasters may cause mass casualties which require surgical interventions, creating a challenge for emergency and hospital healthcare systems, particularly in developing nations. In addition, epidemics such as SARS and COVID19 give rise to large numbers of cases requiring respiratory support and IPPV. Here, mass ventilation provision is required which usually involves non-specialist medical and nursing personnel.

### ***10.2.1 What Is a Disaster?***

Defining disaster from a medical standpoint has been the matter of some debate over the years. Events may be local but catastrophic as in the case of an industrial accident such as the release of methyl isocyanate at Bho Pal in India in 1984 or a terrorist bombing attack as in London in 2007 or may involve a large scale natural catastrophe as in the case of an earthquake, tsunami or volcanic eruption. Overall, any medical definition of disaster involves the number and nature of the casualties and the resources available to deal with them. Thus the definition of a disaster used by the World Health Organization (WHO) is ‘a sudden ecological phenomenon of sufficient magnitude to require external assistance.’ A simpler definition is ‘an event that causes mass injury or illness which overwhelms normal emergency medical responses. This definition particularly applies to artificial ventilation, since it may be required equally for traumatic, infective and toxic causes.

### ***10.2.2 Effects of a Disaster on Medical Care***

Disaster can affect both (1) hospitals in developed nations which are heavily-dependent for their support on essential services such as power, water, gas-supplies and communications and (2) hospitals in developing nations which may have only the most basic facilities available during normal practice, without the effects of a disaster. These are the most vulnerable and are rapidly overwhelmed in the face of mass casualties.

### ***10.2.3 Emergency Ventilation Where Hospital Facilities Are Disrupted***

Providing artificial ventilation in the austere conditions which surround disaster requires planning, equipping and particularly training and familiarity with the ventilators before the disaster has occurred. Ventilators should be chosen that are suitable for disaster conditions where mains power and compressed gas supplies may be absent. Most modern hospital ventilators are not at all suitable for this setting. The technical options for providing mechanical artificial ventilation during and after a disaster depend on the following assumptions:

#### **10.2.3.1 Power Availability**

The absence of normal power supply or means of recharging an internal battery affects both pneumatic ventilators that are partially power-dependent and turbine transport ventilators. This problem can be overcome by using ventilators that are not power dependent or an autonomous ventilator such as CompPac shown in Fig. 10.1 which uses an internal compressor with multiple powering options or by using a compressor to produce compressed air that can be used to drive a portable gas-powered ventilator with oxygen enrichment of the gas in the patient circuit.

#### **10.2.3.2 Oxygen Availability**

In disaster and remote medicine settings, the supply of bottled compressed oxygen may be non-existent or erratic. Oxygen must therefore be used carefully in this situation. The use of the airmix function on PGPV will extend the life of a standard oxygen bottle. Table 7.1 (Chap. 7) gives the capacity of different sizes of available oxygen cylinders and Table 8.1 (Chap. 8) shows typical endurance times for a pneumatic ventilator running on 100% oxygen and airmix. An alternative to using compressed oxygen is to use an electrically-powered oxygen concentrator to enrich air which is used as the driving gas (Box 10.1).

**Fig. 10.1** The Pneupac CompPAC autonomous portable ventilator. This ventilator, originally designed for use in toxic environments has been used widely around the world in difficult circumstances, including military field hospitals. It contains a small internal compressor which compresses filtered ambient air as the driving gas. The compressor can operate from an internal battery, a 28 volt DC or 240 volt AC source, or from compressed oxygen or air if these are available (Photograph courtesy of Smiths Medical International Ltd, Luton, UK)



The 2020 COVID19 outbreak brought the supply and careful use of medical oxygen into sharp focus. The World Health Organisation has issued a guide to the provision of oxygen requirements during a pandemic. (See suggestions for further reading).

#### **Box 10.1 Oxygen Concentrators**

Oxygen concentrators pass compressed air through a substance called Zeolite which has the property of adsorbing nitrogen more than oxygen. Thus if the gas is passed through Zeolite several times it is possible to produce high concentrations of oxygen at a flow of 3–5 l/min. This can be used to provide free flow oxygen or to supplement oxygen to a pneumatic ventilator being

driven by air. Oxygen concentrators require a power source to drive the compressor. This can either be from a battery or from a generator making them suitable for use in difficult environments.

### **10.2.3.3 Problems of Maintenance and Servicing of Ventilators in Remote Locations**

Modern ventilators, particularly the new generation of transport ventilators depend upon the provision of servicing by manufacturers. While servicing of less complex ventilators is possible by training local engineers, in developing countries it may be the case that the ventilator being used in a remote location will be far from the nearest service point. Thus in such situations the use of simple ventilators of known reliability and which can be serviced locally is advisable.

## ***10.2.4 Factors Which Affect the Choice of Ventilator for Use in Disaster Settings***

There are many factors which affect the choice of the most appropriate ventilator to use in disaster circumstances and in remote locations. These include:

- Cost
- Training
- Skills of the operators
- Environment in which the ventilator is to be used (eg urban or rural, desert conditions, high temperatures and humidity).

It is likely that there is no one ventilator which will fit all settings and skill levels. The important thing is to choose a ventilator that is suitable for working in the setting and is understood by the operator with a certain skill level.

In remote locations and disaster medicine electronically-controlled turbine transport ventilators are probably not an option due to their high cost and complexity. Medium range pneumatic ventilators are likely to be the best option. A versatile device such as the Parapac Plus 310 (given as a teaching example in Chap. 7) with its multiple oxygen platform options offers the best option. There are many alternatives at this level of technical sophistication such as the Dräger VN300. The actual choice of ventilator will depend on many factors which are discussed in Appendix B. Complex ventilation modes are not indicated for primary and transport ventilation in difficult circumstances. CMV/demand supplemented by PEEP offers the best solution.

In many circumstances resuscitation ventilators may also provide valuable service in difficult environments. They have the advantage that they are designed to replace bag-valve (BV) ventilation and thus release one operator for other duties if

two person BV ventilation was being used. The hands of the ventilator operator are freed for other tasks while he maintains the essential monitoring of a patient on a ventilator.

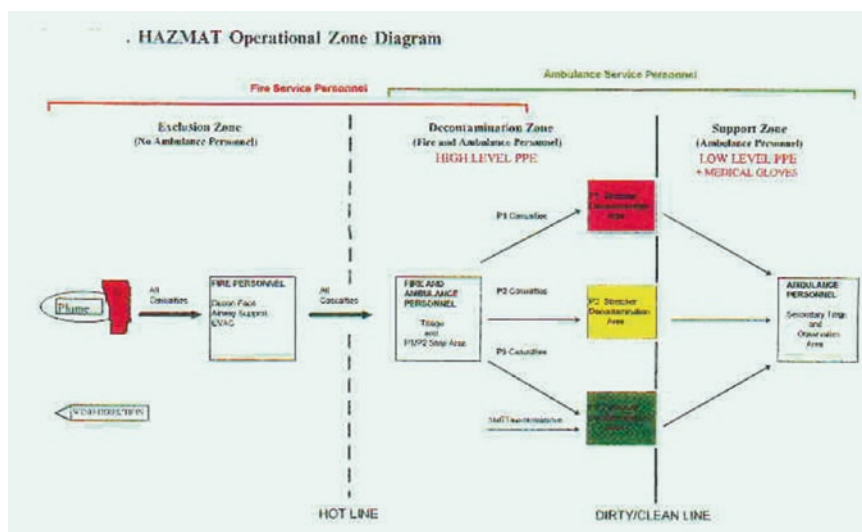
## **10.3 Ventilation in Toxic Environments and Following Exposure to Products of Combustion**

### ***10.3.1 Introduction***

Airway and ventilation care in normal practice or following disasters is provided in an environment that usually presents no risk to the responders. A major exception in the military field is during the course of a battle. However, one area that is common to both military and civil emergency medical responses which is hazardous for the medical responder is the need to provide care for casualties following the release of toxic chemical agents. In the civil area this may be following the accidental release of chemicals or deliberate release by terrorists. For the military the casualties are the result of the use of chemical weapons in battle. In both situations the casualties are at immediate risk from respiratory failure (both type 1 and type 2) and may require urgent ventilator support. If the released chemical agent is persistent (ie it is not dispersed by wind, rain and other natural actions) it will remain in the area of release and possibly on the casualties themselves, presenting a risk to medical responders of becoming secondary casualties. Therefore the provision of artificial ventilation following a toxic agent release will require ventilators that can be used in a contaminated environment, operated by emergency responders who are protected with chemically—resistant suits and filtration respirators (or ‘gas masks’).

### ***10.3.2 Organisation of Emergency Medical Responses Following a Chemical Agent Release: HAZMAT Zones***

Emergency responses following toxic agent release is a special subject that has been the subject of a number of books. The reader is referred to suggestions for further reading. The essential point about management in the civil setting however, is that the areas around any toxic release may be divided into hot, warm and cold zones. A hot zone is defined as the area where the concentration of the release agent is high and presents a direct risk to persons within the zone. Normally only fire and rescue teams equipped with high protection suits and self-contained respirators enter this area to rescue casualties and transfer them to a zone further out called the warm zone. In this zone the risk of chemical contamination comes from contact with contaminated casualties rather than from direct contact with the chemical agent release. An exception is where the released agent is in the form of a non-persistent gas



**Fig. 10.2** The operational organisation of a hazardous materials (HAZMAT) response

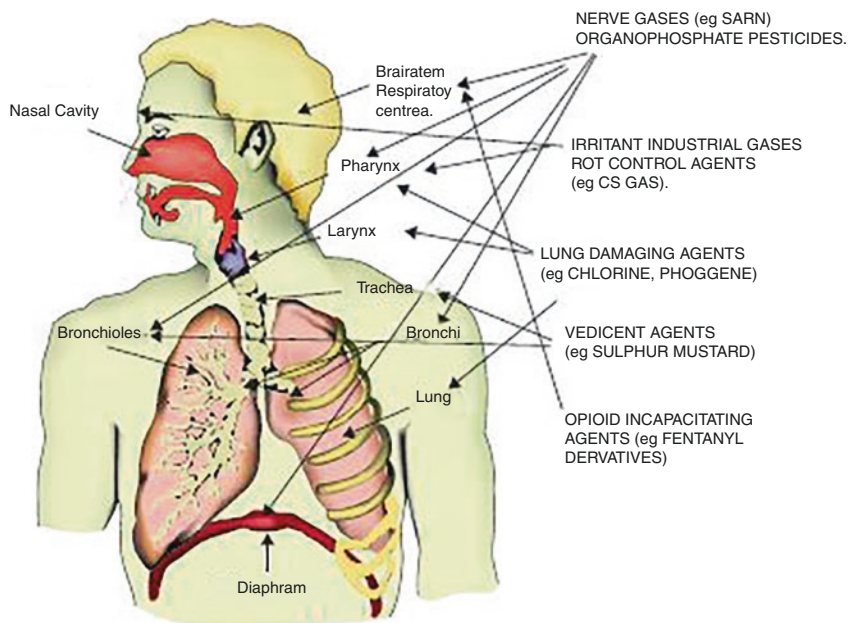
which will form a plume due to wind-dispersal. Here the warm zone is defined by the reduced concentration of the toxic agent in the atmosphere. The important feature of the warm zone is that it is here that vital ventilator care may be provided by emergency responders protected by a lighter suit with a filtration respirator. Finally, the cold zone is defined as the area outside the warm zone where patients have been decontaminated and can be passed into a chain of conventional emergency medical care and evacuation. Figure 10.2 shows the organisation of HAZMAT (hazardous material) response zones.

### 10.3.3 Toxic Respiratory Failure: An Overview

Many toxic chemical agents have an effect on the respiratory systems at all levels of structure and control. It is therefore helpful to have a basic grasp of the actions of chemical agents on the airways, lungs and brain to understand the requirement for artificial ventilation in this extreme environment.

The respiratory system consists of the upper large airways and the lower small airways and alveoli (Chap. 2). All levels of this system are vulnerable to toxic trauma from inhalational chemical agents. These may be classified in terms of the anatomical structures affected as shown in Fig. 10.3. Respiratory failure following exposure to toxic chemical agents has two causes. The first is failure of oxygen to be able to diffuse across the walls of the alveoli into the pulmonary capillaries and thus into the arteries and cells. The second is the failure of the breathing mechanism to move gas in and out of the lungs leading to a build-up of carbon dioxide in the





**Fig. 10.3** Actions of toxic agents on the respiratory system (Reproduced with permission from Baker DJ, *Toxic Trauma—a basic clinical guide* (2nd edition) London, Springer, 2016)

alveoli. These essential types of respiratory failure are known as type 1 and 2 respiratory failure and were discussed in Chap. 4. It should be noted that from toxic causes type 2 respiratory failure has a shorter latency of action (the time taken for the appearance of signs and symptoms) than type 1.

### 10.3.3.1 Irritant Effects on the Respiratory System

The structures of the upper airway including the nasopharynx, larynx, trachea and main bronchi) are susceptible to short latency attack by gases which are relatively water soluble. These include sulphur dioxide, hydrogen chloride and chlorine. The epithelial membranes are affected directly causing rapid inflammation and swelling. Sensory mechanisms are activated with give rise to the classic coughing and choking signs and symptoms.

Due to the high water-solubility and intolerable mucous membrane irritation, prolonged exposure to these gases is unlikely, unless the patient becomes trapped in a toxic environment. If, however there is prolonged exposure or exposure to high concentrations of these highly water-soluble irritant gases, there can be lower airway injury in addition to upper airway injury.

Chlorine dissolves in water less readily and less quickly than hydrogen chloride. When chlorine dissolves in water, it produces hydrochloric acid and hypochlorous



acid. A moderately water soluble irritant gas, like phosgene, produces effects that are similar to but intermediate between the effects of the highly water-soluble and the slightly water-soluble irritant gases. In other words, a moderately water-soluble irritant gas produces both upper and lower airway damage. However, the effects are dose-dependent. Chlorine produces toxic effects on both the upper and lower respiratory tract and the lung tissue. Artificial ventilation may be required in both the primary exposure to chlorine and also after the longer latency effects begin to be apparent, (up to 24h following exposure). Following exposure to high concentrations of gas the latency period is considerably reduced as was seen during the first chlorine gas attacks in 1915 during the First World War.

### 10.3.3.2 Corrosive Effects

Some of the gases that act initially as irritants have further corrosive effects on the structures of the upper airway. Ammonia dissolves in the water covering mucous membranes to form ammonium hydroxide, a strong base that can produce liquefactive necrosis. Hydrogen chloride dissolves in the water covering mucous membranes to form hydrochloric acid, a strong acid that produces coagulative necrosis. Sulphur dioxide has the same effects due to the formation of sulphurous acid. Mustard gas, when inhaled also produces necrosis of the large airways with blockage due to sloughing of ulcerated necrotic tissue. All these agents will produce type 2 respiratory failure as a result of blockage of the airways.

### 10.3.3.3 Effects on the Lower airways and Alveoli

Irritant gases that are slightly water-soluble, such as phosgene and nitrogen dioxide, still produce significant irritation to the upper airways which provides a warning of exposure. Phosgene, which like chlorine was used as a chemical warfare agent in WW 1 produces an initial choking reaction. But the lower solubility of phosgene allows significant amounts to be carried down to the lower airways and alveoli where it causes acute lung injury and toxic pulmonary oedema. These two actions have different latencies, the choking action being almost immediate and incapacitating with PE occurring after 18–24 h and producing a serious life-threatening action. Box 10.2 summarises actions of toxic agents causing respiratory failure.

#### **Box 10.2 Chemical Causes of Acute Respiratory Failure**

##### **1. Nerve agents**

- Central respiratory drive failure
- Peripheral NM paralysis (depolarising as part of the cholinergic syndrome)

(There is Type II respiratory failure in the case of nerve agent—induced ventilatory failure with build up of CO<sub>2</sub> and the production of hypoxia)

Other actions of nerve agents on the airways lead to the exacerbation of respiratory problems. These include:

- Airway blockage from secretions
  - Bronchospasm leading to increased airway resistance
2. Cyanide agents
    - Mitochondrial respiratory failure
  3. Lung damaging agents
    - Initial irritant effects and ‘choking’
    - Possible early type 2 respiratory failure
    - Longer latency production of toxic PE and later ARDS (with associated type 1 respiratory failure)
  4. Vesicant agents
    - Direct effects on the large airways with sloughing and airway blockage
    - Chemical bronchiolitis and small airway constriction
    - Type 2 respiratory failure
    - Also possible toxic pulmonary oedema with type 1 respiratory failure if a dust vector impregnated with mustard agent has been used.

### ***10.3.4 Managing Respiratory Failure in a Toxic Contaminated Zone***

#### **10.3.4.1 Protection of Responders**

The identification and the risks presented by a released toxic agent may not be clear in the initial stages of an incident. Fire and emergency medical personnel responding to such incidents must therefore take appropriate steps to wear suitable personal protective equipment (PPE) to avoid becoming casualties themselves. We saw above that fire and rescue services are equipped with suits and self-contained breathing apparatus that allows entry into any toxic zone with high concentrations of agent whose identity may not have been established. For fire personnel, one of the biggest toxic dangers is carbon monoxide which is not removed by filtration respirators. For this reason they have always used self-contained breathing apparatus which provides its own source of air. In addition filtration respirators should not be used in situations where there is reduced ambient oxygen in the air. Again, this occurs in fires. In contrast to the civilian situation, the military have traditionally taken a different approach to PPE since carbon monoxide is not usually a battlefield hazard and self-contained breathing apparatus is not easily deployed in the field. Thus

military PPE uses relatively lightweight suits with filtration respirators where ambient air is filtered through a cartridge containing activated charcoal mixed with silver salts to remove cyanide. This approach has now been adopted by paramedical and medical teams in many countries for the following reasons:

1. The degree of secondary contamination on patients who have been removed from the hot zone is considerably less than the contamination at the point of release. This means that heavy, self-contained suits with SCBA are not necessary
2. The use of lightweight suits allows essential life support and other medical care to be given early to victims by protected medical responders. Such suits are known as level C suits (Fig. 10.4)

Level C suits are regarded by many emergency medical services in Europe and other parts of the world as being suitable for operation by emergency medical services personnel working in the warm zone to provide essential care to patients awaiting and during decontamination. The weight of the suits and thickness of the gloves allows manoeuvrability and dexterity which permit essential medical operations such as inserting a pharyngeal airway or intubation and providing artificial ventilation.

#### 10.3.4.2 Airway and Ventilation Options in a Toxic Environment

##### Airway

Emergency artificial ventilation must be provided by a ventilation device linked to an airway device. A number of options exist, the use of which depends on the skills of the responders and experience (see Chap. 6). Sometimes a combination of airway and ventilation that would not be regarded as offering the best response can produce a significant improvement in oxygenation because it can be applied quickly. Although the ETT offers the best airway protection, if there are significant delays in placing the tube a worsening of the hypoxia from respiratory failure will result. In general, when wearing PPE in a contaminated zone the LMA offers the most practical way of securing the airway.

##### Ventilation

A number of ventilation options exist which can be used inside a contaminated zone.

- The bag-valve mask device with a chemical filter.

The bag-valve method of artificial ventilation was considered in Chap. 6. The advantages and disadvantages apply particularly for ventilation in a toxic environment where there may be substantial changes to airway resistance and lung compliance. Nevertheless, special versions of the bag valve device have been produced which are suitable for hand ventilation in a contaminated zone.

**Fig. 10.4** Level C protection used by the Paris Emergency Medical Service. (Author's image)



**Fig. 10.5** The AMBU Mk 3 butyl variant bag-valve device with a filtration canister for use in a chemically contaminated environment. (Author's image)



**Fig. 10.6** (a) The PneuPac VR1 (CBRN variant) portable ventilator for use in resuscitation of patients with toxic trauma in a contaminated zone. The butyl rubber patient circuit is fitted with a filter to enable ventilation in chemically contaminated environments. (b) The CompPAC ventilator which uses filtered ambient air as the carrier gas. (Photograph courtesy of Smiths Medical Ltd., Luton, UK)

Figure 10.5 shows the AMBU Mk 3 bag which is made of butyl rubber and draws in air from the surrounding environment through a filtration canister containing activated charcoal. This is identical to the type of canister used in a filtration respirator shown in Fig. 10.4. The bag may be used with either a face mask or a laryngeal mask which are the most suitable airway options for warm zone operations.

- Portable emergency ventilators.

Portable pneumatic ventilators may be used in a toxic environment if the patient is intubated and the device is running on 100% oxygen from a cylinder source. However, this precludes the use of airmix since the ambient air is contaminated. One way of overcoming this problem is to insert a chemical filter between the ventilator and the patient as is the case with the PneuPac VR1 shown in Fig. 10.6. The CompPAC ventilator, mentioned earlier as an example of an autonomous ventilator

was originally designed for use in a contaminated environment. It uses a different approach from the VR1 since air taken into the internal compressor in the ventilator is filtered before entry. The compressed filtered air is used to drive a pneumatic ventilator unit. It is possible on this device to enrich the oxygen content of the gas sent to the patient by adding free flow oxygen at atmospheric pressure to the flow of gas to the patient through the patient circuit producing an inspired oxygen concentration of up to 72%.

Note that the airmix option on the VR1 should not be used for ventilation in a chemically-contaminated environment unless the CBRN filter is included in the patient circuit.

## **10.4 Effects of Inhalation of the Products of Combustion**

### ***10.4.1 Introduction***

Smoke inhalation is a common form of toxic trauma. Damage to the respiratory tract and the lung tissue may be caused by thermal burns due to the transmission of heat by inhaled gases or by the irritant properties of the chemicals contained in smoke itself. Combustion produces a wide range of potential irritants. The composition of smoke varies widely and depends on the nature of the fire, its temperature and what is burning. The considerations for the damage these cause after inhalation are:

- The form of the inhaled substance: particulate or gaseous?
- Chemical properties of the smoke: is it acid or alkaline, and what is the water solubility
- Temperature of inhaled gases which cause burn injury to the respiratory tract.

### ***10.4.2 Asphyxiant Gases***

Smoke contains gases which affect respiration. These may be divided into:

- Active asphyxiants which have a direct toxic action on key systems in the body. These include:
  - Carbon monoxide
  - Hydrogen cyanide
- Passive asphyxiants which produce hypoxia without damage to the lung by type 2 respiratory failure. These include
  - Carbon dioxide
  - Oxygen depletion in the inhaled atmosphere

### 10.4.3 Irritant Gases

Irritant gases are defined as gases which are generated by the combustion of a range of products both natural (eg wood) and synthetic (eg plastics). Combustion may be smouldering, flaming or ventilation-controlled. The irritant gases evolved during combustion and the rate of generation depend on the mode of combustion relative to temperature and ventilation.

Irritant gases can be divided into two groups:

- Inorganic
- Organic

A summary of the properties of common specific irritant products of combustion is shown in Box 10.3.

#### **Box 10.3 Properties of Common Specific Products of Combustion** **Halogen Acids**

**HCl**—This is the most important irritant produced during combustion. Key points in the action of inhaled HCl produced by combustion are (1) the water solubility of HCl means that its effects will predominate in the upper respiratory tract but pulmonary irritation can occur with alveolar damage and pulmonary oedema following exposure to higher concentrations. (2) In relation to phosgene, another pulmonary irritant which can be produced as a product of combustion (*vide infra*) HCl was thought to be the end stage product causing damage at the parenchymal level. However, this was never proven. There is an interesting comparison with chlorine (not a product of combustion) which also acts on the upper respiratory tract as a result of its water solubility.

**HBr**—This is produced as a result of brominated flame retardant compounds or from the combustion of high impact polystyrene.

There are few data available about the toxicity of HBr but its effects are expected to be the same as another halogenated acid, hydrogen fluoride. A notable point about HBr is that exposure to low concentrations (5 ppm) would alert the victim to the potential danger from exposure. However, higher concentrations (200 ppm) produce disabling sensory irritation sufficient to prevent escape from the hazard area. There are recorded cases of death following exposure to 1300–2000 ppm.

**HF**—This halogenated acid can be produced as a product of combustion of fluorine containing polymers eg poly-tetrafluoroethane (PTFE). In conjunction with PFIB there is a severe risk of the production of toxic PE. PFIB was regarded as a potential CW agent at one stage of the Cold War. HF is known to be a powerful sensory irritant. 5 ppm for a one hour exposure causes irritation and corrosion of the mucous membranes of the upper respiratory tract. Volunteer studies have shown lower airway irritancy levels of 0.2–2.9 ppm. Exposure at this concentration gives rise to chest tightness, coughing and



wheezing. At 3–6 ppm these symptoms become more severe. Animal data show that HF is the most potent of the halogenated acid gases but is equivalent to HCl and HBr based upon the endpoint of sensory irritation.

### **Sulphur Dioxide**

This gas can be produced from burning fossil fuels eg coke and from sulphur containing compounds eg vulcanized rubber tyres. It is extremely irritant, particularly in sensitive individuals where severe bronchoconstriction of asthmatic proportions can occur. In known asthmatics a concentration of only 0.4 ppm is sufficient to cause bronchoconstriction.

In addition longer term inhalation of SO<sub>2</sub> for 1–6h has been shown to increase airway resistance and decrease FEV1 results and forced expiratory flow.

Exposure to high concentrations can be fatal due to asphyxiation from blockage of the upper and lower airways.

### **Nitrogen Oxides**

Various forms of nitrogen oxides are commonly present in combustion products. Hence the term NO<sub>x</sub> is used rather than specifying the most commonly encountered compound NO<sub>2</sub> or N<sub>2</sub>O<sub>4</sub>. Any nitrogen containing compound can produce NO<sub>x</sub> depending on the combustion conditions and temperature. NO is formed first and then oxidized to higher oxides.

NO<sub>2</sub> is known to be an oxidant and irritant leading directly to ALI and toxic PE. Hydrolysis to HNO<sub>3</sub> is suspected as a pathophysiological pathway.

Respiratory symptoms are apparent after breathing the gas at a level of 2.5 ppm. Studies have shown increased airway resistance at this level in volunteers.

Toxic PE may start from exposure as low as 2 ppm. Persons who are known asthmatics are more susceptible to the action of nitrogen oxides.

### **Phosphorus Pentoxide**

This is an important product of combustion due its presence in fire-retardant compounds eg isopropylated triphenyl phosphate compounds in polyurethane foam. These compounds are widely used in many domestic appliances. The pathophysiological action may be due to the result of the formation of orthophosphoric acid.

Ammonia

Chlorine

Phosgene

- these compounds are not inorganic acid gases but can all be produced by combustion.

### **Organic Irritants**

Combustion produces a number of organic irritant gases such as acrolein and formaldehyde. In addition there are over 100 listed polyaromatic hydrocarbons (PAH) which contain at least two fused benzene rings in the



molecular structure. This group includes substances such as benzopyrene, naphthalene and anthracene. These compounds are known to have longer term carcinogenic properties.

Other compounds including dioxins cause longer term problems. However these compounds do not cause acute lung injury as defined in the text.

Isocyanates such as methyl isocyanate released in the Bhopal disaster in 1984 can have early and late effects on the respiratory system and are produced by combustion of nitrogen containing polymers often found in insulating materials and soft furniture fillings.

#### ***10.4.4 Ventilation for Carbon Monoxide Poisoning***

Carbon monoxide (CO) poisoning is one of the most common conditions which requires ventilator support. Artificial ventilation may be required not only in the immediate emergency management of severe cases but also inside a high pressure oxygen chamber for continuing therapy. Carbon monoxide binds to the oxygen-binding sites in the haemoglobin molecules of erythrocytes. This creates carboxy-haemoglobin and decreases the ability to transport oxygen. When CO binds to any one of the four oxygen-binding sites in any haemoglobin molecule, the oxygen molecules at the other binding sites in the same haemoglobin molecule are bound more tightly and are less likely to be released to hypoxic tissues. Therefore, CO decreases the ability of haemoglobin to both transport and release oxygen. The principle of high pressure oxygen therapy and ventilation if required is to reverse the preferential binding of carbon monoxide to haemoglobin. In severe cases of carbon monoxide poisoning artificial ventilation may therefore be required in a high pressure (hyperbaric) chamber. It is therefore important to understand the possible variations in the performance of portable ventilators in these conditions.

### **10.5 Mechanical Artificial Ventilation in Hyper- and Hypobaric Environments**

Artificial ventilation usually takes place in settings where the surrounding atmospheric pressure is normal. However there are a number of circumstances where AV is required where the surrounding pressure may be higher or lower than normal. These conditions have an effect on both the practice of ventilation and the delivery of gases and also on the performance of ventilators themselves, particularly if they are pneumatically powered and controlled.

Familiar high pressure environments include hyperbaric chambers for high pressure oxygen therapy and the pressurised cabins of aircraft.

Low pressure environments are most familiar in the setting of high altitude medicine. The pressurised cabin of an aircraft is in fact lower than normal atmospheric pressure but is considerably higher than that of the surrounding atmosphere at high altitudes. The degree of pressurisation is equivalent to atmospheric pressure at an altitude of 8000 feet.

### ***10.5.1 Hyperbaric Oxygen Chambers***

High pressure oxygen is used for the treatment of carbon monoxide poisoning by reversing the preferential combination of CO with haemoglobin and replacing it with oxygen. This form of therapy is reserved for extreme cases.

Severe cases of carbon monoxide poisoning may require ventilation before they enter the chamber and will do so during therapy. Portable ventilators working in these conditions will require recalibration and the manufacturer's advice should be consulted.

### ***10.5.2 Decompression Chambers***

Too rapid a rise to the surface by divers working at extreme depths may result in decompression sickness. This condition is where nitrogen which has dissolved in the blood at high inspired pressures is released as gas due to its solubility characteristics and causes potentially serious neurological problems due to blockage of blood flow in small vessels in the nervous system. The treatment of decompression sickness is to place the patient in a high pressure air chamber and compress the ambient atmosphere to simulate a return to the depth at which he was working. The gaseous nitrogen in the blood then dissolves again. By lowering the chamber pressure gradually, in line with the published diving tables, the patient can be brought back to normal atmospheric pressure without the formation of bubbles of nitrogen.

For severe cases of decompression sickness where ventilation is required, portable ventilators must therefore be able to operate in a high pressure air environment. Again, the manufacturer's advice should be sought.

### ***10.5.3 Problems in Ventilating in a High Pressure Environment***

The potential problems associated with the operation of portable ventilators in a high pressure environment may be summarized as follows:

- (1) The function and calibration of pneumatic components in the case of a pneumatic ventilator

Gas-powered ventilators are designed to operate at normal atmospheric pressure. Higher pressures cause the density of the driving gas to increase and to alter the characteristics of the flow of gas in the pneumatic components. Normally such ventilators will continue to work but recalibration is required before relying on the frequency and tidal volume settings on the controls.

- (2) Whether an electronically-controlled turbine portable ventilator can be safely used in the high pressure oxygen environment since there may be fire risks.
- (3) potential risks from oxygen toxicity to operators from being in a high  $\text{FiO}_2$  surrounding for too long

### ***10.5.4 Artificial Ventilation in Hypobaric Environments***

Ventilation may be required in settings where the surrounding pressure is less than that of the atmosphere at sea level. This is the case in (1) ventilation in aircraft flying at high altitudes and (2) emergency care at high altitude on mountains.

#### **10.5.4.1 Physiological Considerations for Hypobaric Ventilation**

It is well-known that the atmospheric pressure drops with altitude and this is the basis for the operation of the altimeter in an aircraft. Subsonic commercial aircraft fly at a maximum altitude of 13,100 m. At this level the atmospheric pressure around the aircraft has dropped from 760 mm at sea level to about 110 mm. This contains a partial pressure of oxygen of only 23 mm compared with 150 mm at sea level. This level of oxygen is not sufficient to sustain life.

Experience from mountain climbing shows that man can live comfortably at an altitude of 2500 m where the atmospheric pressure is 510 mm and the  $\text{pO}_2$  is 107 mm. It is worth noting that about 140 million people around the world live permanently at altitudes greater than 2500 m and it is also possible that an equal number visit these altitudes each year on a short term basis to which they are able to acclimatize.

Thus, commercial aircraft overcome the problem of a low surrounding atmospheric pressure (which would be transmitted inside the cabin) by compressing air entering the aircraft to a pressure inside the cabin equivalent to being on a mountain at 2438 m (8000 feet) (the typical cabin pressure ranges between the equivalent of being at 1500 and 2430 m as the aircraft gains altitude). This pressurisation provides a cabin  $\text{PO}_2$  of 118 mmHg. Thus passengers in an aircraft are in fact breathing a hypoxic mixture compared with sea level. However, the accompanying mild hypoxaemia is usually well tolerated by passengers who have a saturation of greater than 90%. Passengers who have respiratory or heart conditions can receive free flow oxygen through a face mask at the cabin pressure which will provide adequate oxygenation.

### 10.5.4.2 The Performance of Ventilators Inside an Aircraft

Ventilation is frequently required for long-distance transportation of patients by air. Planning and logistics for air medical evacuation are part of transport ventilation and were considered in Chap. 8. One important point is that the type of ventilator used in such transportation may be affected by the sub-atmospheric pressure inside the cabin. Because the density of the air is lower at 8000 m the performance of pneumatic ventilators in particular may be affected. This is because they depend for their operation on the characteristics of gases flowing through small orifices as in the case of the pneumatic oscillator (see Chap. 7). Thus the calibration of the ventilator being used in an aircraft may deviate from operation at sea level. In addition to problems of the operation of the ventilator low surrounding pressure will also cause the expansion of fixed gas volumes, for example the cuff of an endotracheal tube or gas contained in a pneumothorax.

The performance of pneumatic ventilators at simulated high altitude has been studied by Flynn and Singh (2008). These authors studied the performance of three commonly used transport ventilators (Dräger Oxylog 1000 and 2000 which are pneumatic in operation and the Dräger 3000 which is an electronically controlled turbine device). The ventilators were assessed in a hypobaric chamber at pressures equivalent to altitudes of sea level and 3048 m. Each ventilator was tested delivering 100% oxygen or 60% on air mix against lung models that simulated (1) normal lungs (2) ARDS with low compliance and (3) asthma with high airway resistance. The authors found that for the Oxylog 1000 the  $V_t$  increased over the altitude range by 68% and the frequency decreased by 28%. For the Oxylog 2000 the  $V_t$  increase was 29% with no change in frequency. For the Dräger 3000, which is a more sophisticated ventilator used by specialists there were no changes.

This study underlines the importance of knowing how any particular portable ventilator may be affected by low ambient pressure. For pneumatic ventilators it is a reasonable assumption that the  $V_t$  will increase while the frequency will decrease. This should be checked against the manufacturers' literature however.

## 10.6 Conclusions

Artificial ventilation following respiratory failure may be required in circumstances that are very different from conventional hospital or prehospital emergency settings. These may be termed difficult and extreme environments.

Such environments include:

- Natural and man-made disasters where the normal medical infrastructure is severely disrupted
- Toxic environments, following the accidental or deliberate release of toxic chemicals or chemical warfare agents or following exposure to smoke and products of combustion

- Hyperbaric and hypobaric conditions such as high pressure oxygen chambers for carbon monoxide poisoning treatment or inside the cabin of a commercial aircraft flying at high altitude
- In everyday medical practice in remote parts of the world where medical supplies and support may be strictly limited

In all these environments the operation of portable ventilators is possible but special requirements such as the provision of oxygen, protection of responders and potential changes in the operation of the ventilator itself should be understood by operators.

## Suggestions for Further Reading

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# Chapter 11

## Artificial Ventilation in the Intensive Care

### Unit: An Overview



## 11.1 Introduction

Previous chapters have described the function and use of portable emergency and transport ventilators which can be used by non-specialist medical, paramedical and nursing personnel in a variety of locations, both inside and outside the hospital.

Respiratory care in the intensive care unit (ICU) has increasingly become the domain of highly specialised physicians, nurses and, in some countries respiratory therapists. However, the COVID 19 outbreak has brought public awareness of ICU care, particularly respiratory support to the fore and has revealed a gap in the understanding of the essential purpose of the ICU in providing respiratory and other support to patients who are critically ill and would be unlikely to survive unless admitted for special care.

Chapter 1 noted how emergency and transport ventilation have developed separately from ICU care over the past decades with the development of increasingly sophisticated ventilators in the ICU. The result has been that the ventilators currently used in the ICU have become highly complex electronically controlled devices which are designed to interact as closely as possible with the patient's respiratory requirements.

Although users of portable ventilators are unlikely to be asked to manage an ICU machine, there is an increasing need for awareness and understanding of these, particularly since, at the time of writing ward staff are being required and rapidly trained to take on HDU and ICU care for mass ventilation as a result of the COVID 19 outbreak.

This chapter will cover:

1. An introduction to the development and current practice of intensive care medicine: what is intensive care?

2. Ventilation modes provided in intensive care and how these relate to modes used in portable ventilators
3. Examples of the development of intensive care ventilators
4. Acute lung injury and acute respiratory distress syndrome (ARDS): an overview for the non-intensivist
5. The lessons learned from the COVID 19 pandemic for the management of ARDS

## 11.2 What Is Intensive Care?

Intensive care may be defined as the provision of medical and nursing support in a special hospital unit to patients who are seriously ill from a number of causes and who would be unlikely to survive unless treated with a variety of specialised measures.

Essentially intensive care involves the provision of:

1. Advanced respiratory support
2. Detailed monitoring of lung and heart function
3. Haemodynamic support
4. Treatment of sepsis and multi-organ failure
5. The management of renal failure
6. Provision of artificial nutrition
7. Management of misfunction of the coagulation system and prevention of thrombotic complications

Conditions that require intensive care include:

1. Trauma following major surgery
2. Infections
  - Viral
  - Bacterial
  - Overwhelming sepsis
3. Multi-organ failure
4. ARDS from multiple causes

Within the scope of this text we shall discuss the respiratory aspects of intensive care where the lungs are affected, either by a primary infection or by conditions affecting the pulmonary capillaries and the ability to transmit oxygen into the blood stream. In many cases, intensive care involves management of a ventilated patient who has other life threatening conditions and requires support for other organ systems.

## 11.3 Respiratory Intensive Care

Respiratory failure was addressed in Chap. 4 of this book and could be classified simply as type 1 or type 2 failure depending on whether hypoxia was caused by a failure of oxygen to diffuse into the pulmonary capillaries from the alveoli or was due to a failure of moving gas in and out of the alveoli leading to a build up of CO<sub>2</sub>. In the ICU conditions requiring respiratory support are primarily those which cause type 1 failure and include severe pneumonia and the many conditions leading to the onset of ARDS (see below). In these conditions the characteristics of the lungs are markedly altered with a decrease in compliance and an increase in airway resistance. Artificial ventilation in ICU patients therefore requires a more complex approach than emergency ventilation, which is essentially adaptive. This is because the lung tissue itself is damaged and therefore requires careful ventilation in order not to cause further damage often referred to as VILI (ventilator induced lung injury). This approach to ventilation has been understood gradually over the past 25 years and has led to the development of complex modes of ventilation provided in the ICU.

### *11.3.1 Understanding the Modes of Ventilation on an ICU Ventilator*

1. The application of modern sensor and microchip technology to ventilation in the intensive care unit has produced sophisticated devices that can respond to the patient's own breathing efforts. In some cases the ventilator can adapt on a breath by breath basis detecting changes in the patients lung characteristics of stiffness and resistance. Ventilation in the ICU setting has thus become a complex means of supporting the patients own respiratory efforts, no matter how slight.
2. The result is an interaction between the patient and ventilator which allows greater comfort for a patient being ventilated over long periods of time. Unlike earlier forms of ICU ventilation the patient does not require sedation or paralysis to be able to adapt to the ventilator.
3. ICU ventilators now have a wide range of modes (forms of ventilation) which cover both volume and pressure targeted ventilation and sometimes both. Although these 'modes' are supposed to be standardised, manufacturers often produce subtle variations for commercial purposes with an often trademarked name (eg Respiration Bi PAP™, which is a partially ventilator-supported version of bilevel continuous airway pressure support, Bi-PAP, but which is not Bi-PAP as usually defined).
4. In the ICU, a number of independent variables (such as inspiratory pressure limit, flow rate and inspiratory time) can now be set by the user to produce one specific dependent variable, tidal volume. Thus in the ICU, because of the con-



trolled circumstances and the sophisticated equipment available (which can only be operated in a controlled environment with piped gas and mains electricity supplies available) ventilation can be tailored to the patient's specific requirements with multiple adjustments by the physician and a readout of the results in terms of oxygenation and a graphical display of the lung characteristics. This can only be done in a patient who requires long term, non-emergency ventilation for life-threatening hypoxia.

5. A full range of alarms and monitoring is available on modern ICU ventilators as well as a real-time screen graphical presentation which shows changes in the volume of gas entering the lungs against the pressure applied
6. Modern ICU ventilators are complex, expensive and essentially non-portable. They do not respond well to being moved around and invariably require a mains electricity supply and piped oxygen for their operation.
7. ICU ventilators cannot usually be used easily in the transport of ventilator-dependent patients from one fixed location to another. As discussed in Chap. 8 this may be only a short distance with hospital, from the ICU to imaging facilities or it may be thousands of miles when a patient has to be moved from an ICU in one country to another. In transport ventilation, a device is required that has been designed for the purpose. The problem for hospital ventilator users however is the selection of a mode that is available on a transport ventilator to match the adaptive ventilation that has been provided in the hospital ICU. Often this will require a degree of sedation for the patient who must be placed on a mode that is safer during transport but which will be less adaptive than the modern ICU modes.
8. Some transport ventilators incorporate modes usually found in ICU ventilators, particularly in terms of the pressure support. These devices are power-dependent and have a limited battery life. They often incorporate visual displays which are themselves a drain on the power supply. In effect, these new-generation transport ventilators are miniature ICU ventilators. Although in theory adaptive, they are often of variable ruggedness and are easily damaged in the circumstances of transport ventilation. However, they represent an attempt by manufacturers to present hospital medical personnel with a smaller version of the ICU ventilator with which they are familiar. This is a different approach to transport ventilation from that of the practitioner who has ventilated a patient during a life-threatening emergency and then has to transfer the initially-stabilized patient to hospital care.

### ***11.3.2 A Comparison of ICU and Emergency Ventilation Modes***

The term 'mode' when used in artificial ventilation refers to the way that gas from the ventilator affects the patient's airways and lungs in terms of pressure and volume changes. All gas delivered to the patient produces changes in both these

parameters which can be seen on ICU ventilators (and some transport ventilators as a pressure volume loop LCD display).

There is often much confusion about the modes of ventilation delivered by ICU and EV and how these inter-relate. This section attempts to clarify the situation starting from the standpoint of ICU ventilation where the most complex interactive modes are used. In other situations where artificial ventilation is required, as in EV or during general anaesthesia simpler modes are required since the lungs themselves function normally.

### ***11.3.3 Volume and Pressure Control of Ventilation: Definitions***

As noted in Chap. 6 artificial ventilation can be divided into (1) volume-targeted ventilation-sometimes called volume controlled or volume cycled ventilation and (2) pressure targeted ventilation-also known as pressure limited ventilation. In the case of some ICU ventilators dual control can be used where both pressure and volume targeting can be used together.

Volume and pressure targeted ventilation can be defined by independent and dependent variables of ventilation. An independent variable in ventilation is that set on the ventilator by the operator. A dependent variable is one that is a result of setting the independent variable. For example, in volume controlled ventilation the independent variable (ie that set by the operator) is volume. The dependent variables are pressure and flow. In pressure controlled ventilation, the independent variable is pressure and the dependent variables are volume and flow.

In addition to volume and pressure-targeted ventilation another important group of ventilation modes exists, namely pressure supported ventilation where the ventilator interacts with the patient's own respiratory efforts in partial respiratory failure.

### ***11.3.4 Volume Controlled Modes***

In volume controlled modes, often found in emergency ventilation the ventilator will deliver a preset tidal volume (an independent variable set by the medical responder). To achieve the preset tidal volume with stiff lungs which do not expand easily or increased airway resistance the pressure generated by the ventilator must rise to a level sufficient to provide the desired tidal volume but which is limited by a predetermined safety limit set by the operator.

In volume control, tidal volumes are guaranteed. However, in the ICU where the lung mechanics may be chronically poor, with volume control the airway pressure may rise to unacceptable levels which may cause discomfort to the patient and possible damage to the lung tissue. In this situation, over sustained periods of time it is usual to ventilate with a more complex pressure targeted or limited mode controlled by feedback sensors that measure lung compliance and airway resistance on a real time basis.

### ***11.3.5 Pressure Controlled Modes***

In this form of controlled ventilation the medical responder controls tidal volume only indirectly. A given pressure limit is set and cannot be exceeded. During inspiration from the ventilator, as air is pushed into the lungs the airway pressure rises and rapidly reaches the pressure control level. The preset pressure is maintained for the duration of the inspiration. In the ICU form of pressure control, the pressure limit, frequency and inspiratory time are operator preset (ie are independent variables). Tidal volume achieved is thus a function of these independent variables.

If the lung mechanics are normal and the lungs are distensible with normal (low) airway pressures the pressure controlled ventilator will be able to deliver more gas to the lungs than with high airway resistance and low compliance.

#### **11.3.5.1 Advantages of Pressure Control Ventilation**

Pressure controlled ventilation guarantees that the airway pressure will not exceed the preset level. This reduces the risk of barotrauma and other risks of high intrathoracic pressure such as diminished venous return to the heart are minimised.

#### **11.3.5.2 Disadvantages of Pressure Control Ventilation**

In a patient with unstable or changing lung mechanics due to injury or disease the tidal volume cannot be delivered consistently. With increased resistance and decreasing compliance the  $V_t$  may drop (along with minute volume) to dangerously low levels while the ventilator may apparently continue to work and cycle normally.

The development of modern ICU ventilators has taken into account changing lung mechanics with the development of complex modes of adaptive pressure control ventilation.

## **11.4 Commonly Used Supportive Ventilation Modes in Hospital Practice**

With the development of microprocessor-controlled ICU ventilators with constant sensor feedback, many variations of volume and pressure targeted ventilation have been developed. What these have in common is that they essentially provide support for a patient who is not in complete respiratory failure and who is able to contribute to the work of breathing. This is a different situation from emergency ventilation required for respiratory arrest. The range of ventilation modes available on modern ICU ventilators is extensive and many of the options are highly specialised. They are detailed in Appendix E for the interested general reader. It may be noted that although at present many of the adaptive ventilation modes appear remote from

those used in emergency and transport ventilation it is likely that some of the options for interactive ventilation will be incorporated into prehospital ventilation in the future, with the aim of reducing potential damage to the lungs from IPPV. These lessons were learned over 20 years ago in the ICU and will certainly be applied to emergency ventilation in the future as the realisation of potential harm from over-ventilation becomes more wide spread.

## **11.5 Non-Ventilatory Airway Pressure Support: CPAP and Bi-PAP**

Essentially CPAP (Continuous Positive Airway Pressure) and Bi-PAP (Bilevel positive airway pressure) provide the same thing, namely a positive pressure airway support to patients who still have respiratory drive and are therefore not in respiratory failure. In other words they are still able to breathe but require positive pressure support to improve the efficiency of gas exchange in the lungs. This is thought to be due to an increase in functional residual capacity by opening up the alveoli.

The difference between CPAP and Bi-PAP is that with CPAP there is a continuous applied preset positive pressure during both inspiration and expiration whereas with Bi-PAP the applied pressure during inspiration and expiration is different. CPAP can be provided simply by a flow of oxygen through specialised circuit (as with the Parapac Plus system) whereas with Bi-PAP the different pressures must be provided by an electronically controlled device that detects when the patient is moving between inspiration and expiration.

Both systems can improve oxygenation in the blood in patients who are still breathing but the Bi-PAP devices are said to be more comfortable for the patient since the applied pressures in inspiration and expiration are different. As with all ventilatory support in the ICU the key is adaptability with a conscious patient. Of course, providing Bi-PAP requires a power and gas dependent bedside device which is not essentially transportable and is expensive.

## **11.6 An Overview of Ventilators Used in Intensive Care**

The range of ICU ventilators now available on the market and their apparent complexity can be daunting for the non-specialist. This section provides a simple introduction to the development of the modern ICU ventilator and the way that sophisticated ventilation modes are now becoming available outside the ICU setting. A limited number of ventilators is discussed as examples. The interested reader is referred to more detailed texts and also to manufacturers' literature and online presentations for more detailed information. Online video tutorials are particularly effective in showing the simplified control settings and monitoring displays which are now a feature of high specification ventilators.

ICU ventilators were originally essentially large electrically-driven ventilation pumps which could operate for long periods and would produce a preset tidal volume that replaced completely the patient's own respiration. Over the years, the increasing availability of sensors and electronic circuits developed for use in computers led to the development of smaller machines that were designed to analyse the condition of the patient's lungs and to produce IPPV that was as closely adapted as possible to the patient's own respiratory efforts. This was termed adaptive ventilation and was largely designed around pressure support ventilation, both complete or assisted. One important point about the development of the modern ventilators is that whereas patients on long term ventilation had to be heavily sedated and paralysed with non-depolarizing muscle relaxants normally used in general anaesthesia such as cis-atracurium and rocuronium, adaptive ventilation was much better tolerated by patients who required far less sedation. There were also clear advantages in the gradual removing of ventilator support as the patient's condition improved (termed 'weaning' from the ventilator).

In parallel with the use of IPPV in the ICU, there were other developments in the earlier use of CPAP and Bi-PAP and now high flow nasal cannula, as non-ventilatory methods of respiratory support together with the use of non-invasive ventilation where IPPV was delivered to the patient via a mask, eliminating the need for intubation in some cases.

### ***11.6.1 Features of ICU Ventilators: The Development Over the Past 40 Years***

Before 1980 most ICU ventilators were large heavy units such as the Engstrom (see Chap. 1) which were essentially bellows devices activated by a large electric motor which could deliver only a very limited range of volume controlled ventilation. They had the advantage of being robust and reliable for use over long periods but could not provide adaptive supportive ventilation of the type largely used in the ICU today.

### ***11.6.2 The Servo 900C***

The situation changed in 1981 with the introduction by Siemens of their 900 ventilator (Fig. 11.1). This was an advanced ventilator for its time, the Servo 900C had a single-circuit, with a spring-driven bellows that was pneumatically powered and electronically controlled.

The ventilator modes available included volume or pressure control, pressure support and synchronized intermittent mechanical ventilation. The 900C could also



**Fig. 11.1** The Siemens 900 ICU ventilator (1981); an early example of an electronically controlled ventilator. (Author's image)

be used to aid ventilation with positive end-expiratory pressure (PEEP), continuous positive airway pressure (CPAP), and negative end-expiratory pressure (NEEP).

The Servo 900C was revolutionary for its time and could control a number of parameters, such as airway pressure, tidal volume, and respiratory rate. Add-on units could measure carbon dioxide production and ventilator parameters. Alarms could be set at desired values for the monitored parameters. The device was used in both the operating theatre and ICU. However, all the settings and monitoring were done manually with the operator feeding in the required ventilation parameters and adjusting the ventilator as the lung characteristics changed.

### 11.6.3 *Hamilton C6*

The situation by 2020 has changed completely with the use of highly sophisticated computer controlled ventilators such as the Hamilton C6 which required only minimal setting by the operator and which were then able to provide fully adaptive ventilation to the patient based upon appropriate algorithms for lung conditions such as ARDS (see below) that had been worked out early in 2000s. It was no longer necessary therefore for the ventilator operator to adjust the ventilation parameters as lung characteristics changed. The ventilator had in effect become an 'autopilot' in the modern aviation sense where most of the flying of a modern aircraft is programmed by the pilot according to the flight plan and the flying is controlled by several computers but monitored by the pilot who can intervene and override the programme manually where necessary.

Overall, computer algorithms have simplified the setting of ICU ventilators compared with the complex manually - set devices of 20 years ago. The progress has been driven by studies of ARDS and the development of optimal PEEP and low  $V_t$  strategies.

The Hamilton C6 ventilator provides a good example of the state of the art approach to computer controlled ventilators (Fig. 11.2). This device has incorporated all available volume and pressure support modes of the ventilation into a combined mode known as Adaptive Support Ventilation. Using sensors for flow, PIP, end tidal  $\text{CO}_2$  and oxygen saturation the ventilator can monitor IPPV to the patient on a breath by breath basis and make small adjustments if the compliance and resistance of the lungs changes. The responses are based upon the recommended settings of the ventilator in a number of recognised conditions such as COAD and ARDS. To set the ventilator, the operator has only to enter the patient's weight and height, the target acceptable end tidal  $\text{CO}_2$  and the known condition of the lungs when commencing ventilation. The ventilator then operates using a system called Intellivent

**Fig. 11.2** The Hamilton C6 advanced ICU ventilator 2017. An example of a modern computer controlled ventilator which uses artificial intelligence to provide the ventilation most adapted to the patient's requirements while providing extensive information to the operator about airway and lung mechanics changes on a real-time basis. (Photograph courtesy of Hamilton AG, Switzerland)





**Adaptive Support Ventilation.** For the operator it provides real time graphic displays of all aspects of the evolving respiratory mechanics and the current values of ventilation parameters. The display can be altered to provide information which is of use to the nursing staff, such as the need to provide airway clearance. The essence of the Hamilton approach and that other modern ICU ventilators is to provide the least effort of breathing by the patient and aid a smooth return to spontaneous ventilation during the period when the patient is weaned from the ventilator.

#### **11.6.4 *Hersill Vitae 40***

In line with the new intelligent automated ICU ventilation many of the features of the technology have now been incorporated into portable devices which, although specified as high technology transport ventilators can effectively provide the same level of adaptive ventilation now available in the ICU. The implications of this are (1) that complex adaptive ventilation can be continued during transport and (2) that lower cost adaptive ventilators can be used for mass pandemic ventilation in high dependency units when the number of available ICU beds becomes saturated.

The technology available in high specification ICU ventilators is now available in ventilators that are portable and can provide such adaptive ventilation previously only available in the ICU. An example is provided by the Hersill Vitae 40 portable ventilator shown in Fig. 11.3.

This ventilator is a sophisticated electronically controlled pneumatic device which uses compressed oxygen provided via a reducing valve at pressures between 2.7 and 6 bar as with pneumatic ventilators described in Chap. 7. However, the control systems are essentially electronic and offer a wide range of both volume and pressure controlled ventilation. The device weighs only 1.4 Kg and its design makes it essentially a transportable intensive care unit ventilator.

The Vitae 40 provides a full range of volume and pressure controlled ventilation modes as well as CPAP and pressure support ventilation. It also provides CPR ventilation in accordance with the ILCOR guidelines with a manual override found on conventional resuscitation ventilators. Like high specification ICU ventilators there are graphic displays for ventilation curves, trends and parameters as well as capnography and the usual alarm systems.

The interest of this type of ventilator to the non-specialist reader is that adaptive ventilation is moving out of the ICU and it is likely that it will be more widely - used in general emergency and transport ventilation within the next few years. Although the manufacturers' describe the Vitae 40 as an advanced emergency and transport ventilator, its specifications make it suitable for use in adaptive ventilation in the ICU. Because of its relative simplicity of operation it could be used in situations such as the COVID 19 pandemic where high specification ICU ventilators are in limited supply. Ventilators of this type will have an increasing role in the current pandemic and in future situations where mass ventilation may be required.





**Fig. 11.3** The Hersill Vitae 40 ventilator. A compact ( $227 \times 125 \times 65$  mm, weight 1.4 kg) modern electronically-controlled portable ventilator using ICU ventilator technology. It can provide a range of ventilation modes previously only available in the ICU. Originally conceived as an advanced emergency and transport ventilator, it can be used in high dependency units and in ICUs where the ventilator capacity becomes overwhelmed, as in the COVID19 pandemic. (Photographs courtesy of Hersill Medical Devices, Madrid, Spain)

## 11.7 Acute Lung Injury and Acute Respiratory Distress Syndrome

### 11.7.1 Introduction

The COVID 19 pandemic has brought home to the general public the importance of intensive care for a highly contagious respiratory virus, SARSCoV 2. The outbreak is far more extensive than the previous SARS epidemic of 2003 and highlights the need for artificial ventilation for large numbers of cases, which has stretched the ICU capability world wide, both in the provision of medical care and the necessary equipment and essential disposable supplies and importantly, oxygen. The SARSCoV2 virus causes a relatively mild illness in the majority of younger cases but leads to acute respiratory distress syndrome (ARDS) in older patients and those with pre-existing medical conditions. In this group there is a high mortality rate. This section presents the essentials of ARDS and its precursor acute lung injury (ALI) and the lessons that have been learned over the past two decades about the scientific basis for their respiratory care.

### **11.7.2 Definitions**

Adult Respiratory Distress Syndrome was first described by Ashbaugh and his colleagues in 1967. At that time the condition was called Adult Respiratory Distress Syndrome to distinguish it from the previously recognised Respiratory Distress Syndrome (RDS) which occurs in premature neonates. Later the syndrome was renamed as its acute nature became better understood. Initially ARDS was defined by the presence of the following:

1. Acute onset of difficulty in breathing with a high respiration rate and profound dyspnoea
2. Cyanosis (indicating hypoxia) that was difficult to resolve by breathing oxygen
3. Decreased lung compliance
4. The presence of bilateral opacities in the lung field on chest X ray (CXR) that could not be explained by the usual cardiac causes such as heart failure.

ARDS was intensively studied using the relatively recent techniques of evidence-based medicine and was one of the first medical conditions to be considered in this way. Later ARDS was the subject of international co-operation and several conferences and it was realised that ARDS was a severe subset of the previously-recognised acute lung injury (ALI). Both ALI and ARDS are now seen as part of the same clinical spectrum with characteristic bilateral opacities in the CXR. However it was evident that ARDS was more severe and had a higher mortality. ALI and ARDS can be distinguished using the ratio of arterial oxygenation in mm Hg ( $\text{PaO}_2$ ) and the fraction of inspired oxygen ( $\text{FiO}_2$ ). For ALI this is between 300 and 200 mm Hg. For ARDS the ratio is less than 200 mm Hg. This simple definition has its limitations since (1) it does not consider the causes of ALI and ARDS which are multiple (2) it does not quantify the degree of lung opacity nor the response to PEEP which became an essential part of the ventilation treatment strategy. A conference held in Berlin produced a more refined definition. (Ranieri VM et al. JAMA 307:2526, 2012).

Studies on ARDS have been co-ordinated by ARDS Net, a world-wide collaboration which has produced guidelines for respiratory care that have reduced mortality due to a careful ventilation strategy. Lessons learned from these may have important consequences.

### **11.7.3 Causes**

ALI/ARDS is thought to be due to a diffuse and overwhelming inflammatory reaction of the lungs either to direct physical injury as in major trauma or to indirect lung injury related to a systemic process in the body. It has multiple causes that have been recognised for many years. These can be divided into

**Table 11.1** Causes of acute lung injury and acute respiratory distress syndrome

Direct lung injury	Indirect lung injury
Pneumonia	Sepsis and septic shock
Aspiration of acid/gastric contents	Multiple trauma
Air or fat emboli	Acute pancreatitis
Inhalational injury	Cardiopulmonary bypass
Near drowning	Transfusion-related acute lung injury
Pulmonary contusion	Drugs
Post-thrombectomy, post transplantation	Neurogenic pulmonary oedema
SARS and SARSCoV2 (COVID19)	

- (1) direct lung injury from infections such as pneumonia, inhalation of toxic gases, pulmonary contusion, fat emboli, inhalation of gastric contents and near drowning.
- (2) indirect lung damage from sepsis, multiple trauma, acute pancreatitis, cardiopulmonary bypass, ingestion of poisons and certain drugs.

The most frequent indirect causes of ALI/ARDS include sepsis with multiple organ failure, peritonitis and multiple trauma. Sepsis is recognised as being the leading cause since 40% of patients with this condition go on to develop ARDS. There are factors predisposing to ARDS including chronic alcohol abuse. Examples of the causes of ALI/ARDS are given in Table 11.1.

Coronaviruses are now recognised as being a leading infectious cause of ARDS. The SARS coronavirus (known as novel coronavirus or SARS CoV) was first described in 2003 following an outbreak in China. A subsequent analysis of patients admitted to the ICU in that country showed a 25% incidence of progression to ARDS after initial infection, a 37% mortality at 28 days and an overall ICU mortality of 52% after 13 weeks. One third of SARS patients recovered within 14 days. However, most patients had a protracted course with a high mortality rate.

At the time of writing, another coronavirus (SARSCoV2) has swept the planet with a resulting respiratory infection that is less severe than SARS but which is still fatal in over 10% of cases due to progression to ARDS. Due to the extremely large number of reported cases around the world, which stood at more than 25 millions (with over 800,000 fatalities) in August 2020, as a result of unrevealed cases which are infectious in the asymptomatic phase. The number of cases requiring ICU care is proportionally high and has led to serious shortages of ventilators and ancillary equipment together with a shortage of personal protective equipment. The ongoing requirement for mass confinement practised by many countries will have profound consequences for the world economy. ARDS in COVID19 is discussed further in Sect. 11.7.6.

### ***11.7.4 How ARDS Develops: The Key Points***

#### **11.7.4.1 Genetic Factors**

Critical care physicians have noted that patients seem to respond differently to similar predisposing causes of ALI/ARDS. This suggests possible underlying genetic factors. These observations led to intense research into possible genetic predispositions to the conditions. The details of these are outside the scope of this discussion but the interested reader is referred to the suggestions for further reading at the end of this chapter.

At present our knowledge about genetic predisposition may be summarised as follows:

1. there are indications that susceptibility to ALI may be inherited
2. relatively few genes with an altered expression control the complex processes of lung damage and repair
3. Angiotensin converting enzyme (ACE) DD genotype, found in the renin-angiotensin system may have a key role in the pathogenesis of ARDS.
4. RDS in children may be associated with accumulating fatty acid metabolite deposits in patients with enzyme deficiencies and mitochondrial defects may alter the phospholipid component of surfactant and may impair its function in keeping the alveoli patent.

The COVID 19 pandemic has raised questions about why SARSCoV2 causes severe infection and ARDS in some patients and not others. At the time of writing, a large scale investigation of patients contracting COVID19 in its various forms is underway with the hope that, by determining the genetic profile of individual patients there may be indications for treatment.

#### **11.7.4.2 Pathophysiology**

1. The acute or exudative phase of ALI/ADS is characterised by increased permeability of the alveolar/capillary barrier (see Chap. 2) leading to an influx of protein-rich oedema fluid into the terminal airways and the alveoli.
2. Damage to the type 2 cells (see Chap. 2) which make up 10% of the alveolar surface is well-recognised. Injury to these disrupts normal epithelial transport and impairs the removal of oedema fluid from the alveolar space in the majority of patients with ALI/ARDS.
3. The damage to the alveoli is through a pathway of many inflammatory mediators released by macrophages (white cells) responding to the original pulmonary insult. Imbalance between pro-inflammatory and anti-inflammatory mediators may play a key role in the pathogenesis of lung injury. The end result of a vicious circle of inflammatory mediators is alveoli filled with protein-rich oedema fluid,

cells, cellular debris, red blood cells and fibrin hyaline membranes on the damaged basement membrane. This leads to the deposition of fibrin in the alveoli and the interstitial spaces between them which impairs oxygen transport and gives rise to the characteristic lung opacities seen on the chest X ray (CXR) in ARDS cases.

### ***11.7.5 Clinical Presentation***

The initial symptoms of the ALI/ARDS may be non-specific with dyspnoea and dry cough. These worsen with the development of tachypnoea and tachycardia within 12–24 h. Then there is a dramatic increase in the work of breathing and a rapid decrease in oxygenation as revealed by cyanosis. This cannot easily be overcome by breathing oxygen at atmospheric pressure. Auscultation of the lungs reveals high pitched crackles. The patient may initially be agitated but then gradually sinks into lethargy with the worsening respiratory failure. Note that the clinical and radiological findings may lag behind the worsening hypoxaemia so early blood gas analysis is essential.

The hypoxia of ALI/ARDS is due to a combination of:

1. Ventilation-perfusion mismatch
2. Intrapulmonary shunting
3. Impairment of oxygen diffusion (type 1 respiratory failure)
4. Hypoventilation.

The progression to severe hypoxaemia which is unresponsive to breathing oxygen is due to the increase in intrapulmonary shunting and respiratory acidosis from gradual respiratory muscle failure. It should be noted that the clinical findings can sometimes not be distinguished from cardiogenic pulmonary oedema.

The CXR findings are often normal during the first 12–24 h as noted above. Over the next 36 h there is characteristic bilateral interstitial infiltration which progresses to a ground glass opacification. There are often pleural effusions.

Studies of compliance of the lungs during the condition have indicated that there is consolidation of the lungs in patients with ALI/ARDS of pulmonary origin while there is alveolar collapse in cases with extrapulmonary origins of the condition.

### ***11.7.6 Ventilation Strategies***

Effective adaptive IPPV is essential in the management of ALI/ARDS and there has been much research over the years into determining the best way of delivering this without causing VILI. These have centred on the need to avoid further damage to

the alveoli by continuous opening and closing and friction of the collapsed alveolar walls (volutrauma). It became apparent early on that reducing the tidal volume below normal levels provided in IPPV was a key step. A trial by the ARDS Net was stopped after the enrolment of 861 patients since there was clear evidence of lower mortality in patients treated with lower rather than conventional tidal volumes. Since then, application of the low  $V_t$  strategy has been shown to be associated with a reduction in the levels of the inflammatory mediators involved in the development of ALI/ARDS. Protocols have now been developed which combine the use of pressure support measures (CPAP and Bi-PAP) and adaptive pressure support ventilation (Appendix E) for the early stages of respiratory support and gradually reducing the ventilator support for the patient during the recovery period (weaning). A typical modern ventilation support protocol used for low tidal volume ventilation of patients with ARDS has been developed by the University of California, San Francisco (Liu and Gropper 2015—see suggestions for further reading).

Increasingly veno-venous extracorporeal membrane oxygenation (ECMO) is used in severe ARDS. Venous blood is continuously drained from the body, oxygenated and cleared of  $\text{CO}_2$  in an artificial lung, and then returned to another large vein. The technique artificially supports the body's respiratory requirements and obviates the need for high ventilator settings with their injurious effects.

### ***11.7.7 COVID19: A Special Case of Artificial Ventilation for ARDS***

The COVID19 pandemic has provided much information about ARDS that appears to be caused by a dual attack via the lungs and the pulmonary capillaries, linking the two major routes for the generation of ARDS discussed in Sect. 11.7.3. ARDS in COVID 19 has been reviewed by Marini and Gattinoni (2020) who have hypothesised that there are two presentations (phenotypes) of severe cases in the ICU following infection with SARS-CoV2. These depend on the degree of compliance retained by the lung and are termed type L, where the compliance is high (L stands for low elastance which is the reciprocal of compliance) and type H where the compliance is low. The reasoning is as follows. Shortly after the onset of respiratory distress from COVID 19 the authors found that patients initially retain good compliance despite poor oxygenation. CT scan at this stage has a 'ground glass' appearance which is characteristic of interstitial rather than alveolar oedema. Many patients stabilise at this Type L phase of COVID 19 and accept higher tidal volumes (7–8 ml/Kg) than is usually used in lung protective strategies. With a compliance value of 50 ml/cm  $\text{H}_2\text{O}$  and a PEEP setting of 10 cm  $\text{H}_2\text{O}$  there is a plateau pressure of 21 cm and driving pressure of 11 cm  $\text{H}_2\text{O}$  which are well below the currently accepted thresholds for preventing ventilator-induced lung injury (30 and 15 cm  $\text{H}_2\text{O}$  respectively). However, some patients go on to develop type H COVID19 where the compliance is reduced and a lung protective strategy with lower  $V_t$  (6 ml/

KG) and higher PEEP. This corresponds to the Open Lung strategy of ventilation of Lachmann (Reis et al. 2008—see suggestions for further reading).

At the time of writing, there is a lively debate about whether the type L and H phenotypes are recognisable or whether there is a spectrum of clinical presentations of severe COVID 19 which encompass a range of different forms of ARDS. In particular, Ziehr et al. (2020) have queried the hypothesis of Marini and Gattinoni since it was based upon a limited series of patients. As analysis of the many thousands of COVID 19 cases which have required mechanical ventilation in the ICU around the world progresses, the picture of ARDS in COVID 19 may become clearer. What is apparent from studies so far is that SARSCoV2 is unusual in the way it causes ARDS which can originate from either the gas or vascular side of the alveolus. Although COVID19 is spread by the inhalational route and the initial attack is on the type 2 alveolar cells there is a notable vascular insult which leads to thrombo embolic complications in the ICU and also to renal failure. Marini and Gattinoni argue that COVID 19 is a systemic disease that primarily injures the vascular endothelium and that inappropriate management may lead to multiorgan failure even in younger patients and those not having a pre-existing medical condition.

The most interesting point about what is known so far about the way SARSCoV2 leads to ARDS is that it appears to bridge the two main groups of insults, both respiratory and vascular in origin which cause the condition.

For the non-ICU specialist the debate about COVID 19 respiratory management may have implications for the way that respiratory distress is managed in the future, particularly in the pre hospital emergency setting.

## 11.8 Conclusions

The COVID19 outbreak has fuelled a rapidly expanding interest in the ICU ventilation and respiratory support among non-ICU specialists and the general public alike.

This chapter has provided an introduction to the development and current practice of intensive care medicine for readers who may not have been involved with this specialised area of medicine. Within the scope of this book, emphasis has been placed upon respiratory care in the ICU and how it relates with the management of respiratory failure described elsewhere. It should be emphasised that respiratory care is only one part of the often complex process of providing intensive care.

Intensive care respiratory support involves both non-ventilatory and ventilatory care. Intensive care ventilators have become increasingly sophisticated compared with emergency and transport ventilators. The ventilation modes provided in intensive care are essentially adaptive and designed to support what remains of the patient's own breathing efforts. These are often complex but are built upon the basic principles of IPPV which are described in earlier chapters.

Much of intensive care medicine is devoted to the management of ARDS. The principles of respiratory management of ARDS and the importance of protecting fragile and often damaged lung tissue have been the subject of intense ICU research for a number of years and have resulted in improving outcomes for what previously had been a condition that was associated with a very high mortality. The COVID-19 pandemic has provided new insights into ARDS which will have important consequences for ICU practice in the future.

## Suggestions for Further Reading

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# Chapter 12

## Mass Ventilation



### 12.1 Introduction

This book has considered the indications, application and problems of the provision or artificial ventilation from the standpoint of the management of individual cases of respiratory failure. As we have seen, this may be in the hospital ICU where continuing adaptive ventilation is provided by specialist physicians and nurses using sophisticated electronic ventilators or in the emergency and transport setting where less complex ventilators are used by non-specialist personnel. Both these settings are part of everyday medical practice which provides a scale of equipment and hospital beds that are appropriate for the normal incidence of cases of respiratory failure.

However, in parallel with this normal practice there is a recognised continuing risk of the sudden presentation of large numbers of cases requiring artificial ventilation for respiratory support which raises the question of the provision of mass ventilation. Mass ventilation may be defined as the simultaneous ventilation of a surge of patients in varying degrees of respiratory failure who would overwhelm the normal ventilation capability of a medical service. This is a situation which applies equally to developed and to developing countries. In the latter case, the situation is rendered more difficult by the often basic levels of skills and equipment that are available, even for everyday practice.

Over the past 20 or so years there has been a need for mass ventilation for respiratory epidemics such as the SARS outbreak in 2003, and most notably the COVID19 pandemic in 2020 caused by the novel coronavirus SARS-CoV2. In addition there is a continuing risk of mass respiratory casualties following the release, either accidental or deliberate of toxic chemicals. This was underlined by the Bhopal incident in India in 1984 which led to many thousands of casualties with toxic pulmonary oedema and by the terrorist sarin attacks in Tokyo in 1995.

For almost a decade there has been continuing discussion about how best to plan and manage the ventilation requirements for mass ventilation from the standpoints of who should be involved and what are the best ventilator options. The provision of mass ventilation will involve the use of medical, nursing and paramedical personnel who are not ventilation specialists and who will be required to operate with often minimal expert supervision in special units for the provision of mass ventilation using equipment that is more basic than that found in the ICU. Careful planning, training and familiarity with the ventilators to be used, preferably from experience gathered in normal single ventilation practice is essential. In this chapter we consider the recognised characteristics of the best equipment options from a general standpoint. The characteristics of suitable ventilators currently available on the market are presented in Appendix D.

## 12.2 Mass Ventilation: The Scale of the Problem

Previous flu pandemics have caused the deaths of millions of persons world-wide as a result of respiratory failure, usually due to secondary pneumonia. In this situation, respiratory support by artificial ventilation can be lifesaving. However, at the time of the last flu pandemic in 1918–1919 mechanical artificial ventilation was not available. Over a century later the next pandemic, COVID19 was the first in history where mass artificial ventilation was employed.

The first modern respiratory epidemic where IPPV was used was not flu but poliomyelitis. During the polio outbreak in Copenhagen in 1952 there were 2830 cases, of which 1235 had a component of paralysis. Respiratory paralysis was seen in 345 patients of whom 70 required total ventilatory support. IPPV from a ventilator was not available at that time and a manual solution was provided quickly using a non-reforming bag with a soda lime absorber which conserved heat and oxygen and provided automatic humidification (Chap. 1).

The important point about the Copenhagen outbreak is that ventilatory support was provided for neuromuscular respiratory failure where the lung tissue was not affected. Infective respiratory pandemics require a more sophisticated approach where, in some cases the ventilator must adapt to lung changes and to the patient's own respiratory efforts (Chap. 11). These situations have led to discussions (driven in particular by the respiratory therapists in the United States) about procurement of suitable equipment for mass ventilation and importantly, who is available to operate it in mass disasters. The debate has been fuelled not only by the COVID19 outbreak which has stretched ICU ventilation capacities throughout the world but also by the continuing threat from terrorism starting with the sarin attacks in Matsumoto and Tokyo in 1994 and 1995.

Overall the following situations will require mass ventilation:

1. Conventional explosion and blast injury: potentially tens to hundreds of casualties, with IPPV required from days to weeks

2. Inhalational chemical exposure: potentially thousands of casualties, with IPPV required for periods of hours to days. Chemical exposure may cause neuromuscular paralysis with airway compromise due to effects on bronchi and secretions
3. Respiratory pandemics: potentially many tens of thousands of casualties, with lung pathology requiring adaptive IPPV. In many cases respiratory support and IPPV will be required from days to weeks.

## **12.3 Who Should Be Involved in Mass Ventilation?**

The organisation of mass ventilation will require different medical and nursing specialties, many of whom will not normally be involved in the daily ventilation of patients. The groups involved and their skill levels are as follows:

### ***12.3.1 Intensive Care Physicians and Nurses, Anaesthesiologists, Respiratory Therapists***

This group is involved in ventilation on a normal daily basis in the ICU and operating theatre and will be involved in planning and training other groups to be able to operate mechanical ventilation at a level less complicated than the ICU in units usually termed ‘high dependency units (HDU).’ Such units may be set up in hospital when the usual ventilation facilities are overwhelmed. In the HDU, transport ventilators may be used that provide essential ventilation support with CMV and demand ventilation (Chap. 8). These ventilators may be used over a short period to provide support for patients who are in partial respiratory failure from early ALI. The use of the HDU allows the ICU ventilators with their extensive range of interactive modes to be reserved for patients with ARDS who often require complex adaptive IPPV (Chap. 11).

### ***12.3.2 Emergency and Other Medical and Paramedical Personnel Working Outside the Hospital Environment in the Ambulance Service***

Some emergency services use specialists such as anaesthesiologists who have an expert knowledge of artificial ventilation as part of their normal work. This is the case in the French emergency medical service (SAMU) and similar services in other parts of Europe. In other circumstances primary care is delivered by paramedical personnel who should be trained to operate basic resuscitation and emergency ventilators.

In the United Kingdom, which has a paramedically-operated ambulance service helicopter emergency services are supported by emergency physicians who are familiar with the operation of portable ventilators. Such personnel will also be used in a training and supervisory role in the HDU and will be able to train non-specialists in the use of transport ventilators. In many countries, specialist military medical personnel will also be available for this task.

### ***12.3.3 Paramedical Personnel***

Paramedical personnel have varying skill levels in airway management and artificial ventilation. This is often the result of different training policies and equipment levels in different ambulance services within a single country.

In some countries the routine use of the bag—valve device is still widespread (e.g. USA). However, there is increased use of portable ventilators by emergency services around the world to avoid problems posed by the use of BV devices (Sect. 5.6.2).

### ***12.3.4 Nursing Personnel***

Many nursing personnel, apart from those employed in the ICU have training and experience in bag-valve and portable mechanical ventilation as a part of their normal practice, e.g. in the emergency room and recovery areas of the operating theatre. These skills provide a very useful nucleus of personnel who can operate portable ventilators in the HDU under physician supervision.

## **12.4 Providing Mass Ventilation: Guidelines and Practicalities**

Following the COVID19, SARS, influenza and Ebola outbreaks of recent years, together with the increasing threat of mass casualties from chemical terrorism, many countries have reviewed options for the provision of mass ventilation.

In the case of the COVID19 pandemic it was evident that mass stockpiling of ventilators had been neglected in some countries over a number of years. Attempts to rapidly-produce ventilators using engineering facilities with little or no experience in the field met with very limited success. There have been a number of published studies on the planning and desirable characteristics of stockpiled portable ventilators to be used for mass ventilation in the HDU and in field hospitals. Some of these are suggested for further reading later.

As an example this section compares the approaches in the United States, France and the United Kingdom towards mass ventilation. Readers are advised to consult published information about stockpiling in other countries as necessary. However, this is sometimes not easily available. The provision of mass ventilation now has a high priority in the nations worst affected by the SARSCoV2 virus.

### *12.4.1 The United States*

In-hospital ventilation in the US is largely controlled by registered respiratory therapists (RRT), a specialty that does not exist in Europe and other parts of the world. RRTs work on the wards and in the ICU but usually not in the ED nor in the prehospital scenarios. They are largely familiar with sophisticated ventilators of the type found in the ICU which are able to adapt to changes in the patient's respiratory system as a result of a disease process. They are not necessarily familiar with emergency ventilation nor with ventilation required as part of general anaesthesia which is provided in the US by nurse anaesthetists and anesthesiologists respectively.

In 2006 the American Association for Respiratory Care (ARRC) studied the requirement and practicalities for stockpiling of ventilators in the event of a mass disaster requiring the ventilation of a large number of patients. Their findings were published in a paper which examined the problems posed by mass ventilation requirements during an avian flu pandemic and assessed the equipment options available to provide a guide for authorities charged with procurement. The main findings and recommendations of the ARRC (2006) paper were:

1. Ventilator reserves must be versatile enough to meet the ventilator demands of a mass casualty and/or pandemic event.
2. Planners should consider standardization of ventilators when practical, in order to simplify (a) training support staff, (b) inventory of support resources (circuits, etc.) and (c) anticipated site of use.
3. Ease of usage and training must be considered at the time of ventilator purchase.
4. Numbers and types of ventilators should reflect the differences in need between disaster response with mass casualties and a pandemic (author's note: at the time this was thought to be most likely due to the H5N1 influenza virus. The events of 2020 have proved otherwise.)
5. Ultimately, there will be just one reserve of ventilators to use in both disaster scenarios. As such the need to add ventilators that have ventilation mode capabilities to support pandemics is paramount.
6. The current US ventilator stockpile should be expanded by 5000 to 10,000 ventilators. This should include approximately 1500 ventilators (1000 adult and 500 pediatric) with the features and capabilities that can support patients with Acute Respiratory Failure.

Later in 2006, another paper appeared (Rubinson L, Branson RD, Pesik N et al, 2006) written by a wide ranging group of respiratory physicians and respiratory

therapists some of whom were part of the US bioterrorism response programme. These authors provided a balanced US view of the problems of mass ventilation both in terms of the equipment options and the problems faced by lack of trained personnel to operate it. Previous papers, notably that from the ARRC had taken the line that only normal full specification ICU hospital ventilators would be adequate for the task of ventilating patients with lung pathology. Robinson et al. took a far more pragmatic approach to the problem and assessed other equipment options. For the first time in the US mass ventilation discussion, the essential role to be played by portable gas or electrically-powered ventilators was recognised.

The main points presented by the Robinson et al. paper are shown in Box 12.1.

### **Box 12.1 Key Perceptions from the 2006 ARRT Assessment of the Requirements for Mass Ventilation (Robinson et al. 2006)**

#### **1. General points**

At the time of publication (2006) the threat of an H5N1 avian flu epidemic was seen as high. At that time 180 persons had been infected by the strain. If the clinical course of these is representative then a large number of patients in a pandemic will develop ARF. (author's note: The COVID19 pandemic has shown that development of ARF and ARDS occurred in about 20% of cases, whereas in the earlier SARS epidemic of 2003 the proportion was about 50%).

Intermittent Positive Pressure Ventilation (IPPV) is the key to treatment of ARF. IPPV is usually provided in-hospital with full specification ICU ventilators that can adapt to the lung changes caused by respiratory infection.

In the US there is a strategic national stockpile (SNS) of ventilators controlled by the Centre for Disease Control and Prevention (CRCP) Atlanta. This amounts to several thousand Impact Univent Eagle 754 and Puritan Bennett LP-10 devices. CRCP is currently considering buying several thousand more devices for mass ventilation. These are likely to be portable ventilators which are adequate for most mass casualty events. However, it is foreseen that the SNS will be overwhelmed by the mass ventilation requirements of an H5N1 influenza pandemic.

The patient need for IPPV is likely to exceed the total number of ventilators currently available.

#### **2. Stockpiling IPPV equipment—features of a potential 'ideal' ventilator**

1. Stockpiled ventilators should be approved by the Federal Drug Administration. Multiple suppliers/manufacturers should be considered to avoid total reliance on one supply and maintenance source.
2. They should be capable of providing adequate ventilation and oxygenation for most adult and paediatric patients with ARF/ARDS

### 3. Alarms and controls

1. Audible alarms are desirable (the minimum should be patient apnoea, high inspiratory pressure, low pressure/disconnection and low driving gas supply)
2. Separate control of respiratory rate and tidal volume, PEEP and  $\text{FiO}_2$  is necessary
3. Devices should be intuitive in use and require minimal training

### 4. Performance

1. There should be efficient use of medical gases. The ideal device should not be dependent on pressurized gas as the sole power supply
2. Devices should be able to operate on battery or a non-AC power source
3. Devices should be able to operate for days to weeks continuously
4. Devices should be inexpensive to purchase and maintain

### 5. Airway management

Only the endotracheal intubation or the laryngeal mask should be used maintain the airway. The authors were against the use of non-invasive ventilation for mass ventilation.

### 6. Evaluation of devices other than full specification (ICU) ventilators for mass ventilation

#### 1. Manual (bag-valve) devices.

The usual advantages and disadvantages of the BVM were considered (cheapness, apparent ease of operation, little training, poor control of ventilation, overventilation and difficulty in maintaining a high oxygen concentration). Importantly however, they also noted the problem of potential damage to the lung by repeated uncontrolled opening and closing of alveoli unless an external PEEP valve can be connected

#### 2. Automatic resuscitators

In the United States there has been interest in stockpiling disposable simple gas powered portable resuscitation ventilators for use in mass ventilation. This was thought to be an attractive option in terms of cost and apparent simplicity of operation. Robinson et al. were strongly against the use of such devices for the following reasons

- They are pressure cycled (author's note: only pressure cycled resuscitators were apparently considered in this review) and therefore cannot guarantee adequate ventilation in infected lungs (see Chap. 7)
- The devices were wasteful of compressed oxygen and of limited endurance
- With a leak in the patient circuit the device may become stuck in inspiration
- The devices are unable to cope with compliance and resistance changes and cycling may occur without any ventilation being delivered
- No alarms are provided

- Experience of the use of one such disposable device (the Vortran) following Hurricane Katrina found it to be severely limited in providing effective AV

3. Automatic transport ventilators (Author's note: this is a US term which includes both emergency and transport ventilators according to the European definition)

The authors regarded the current range of available electrical and pneumatic portable ventilators as a 'reasonable choice for mass ventilation' in terms of price, features and safety and operator skills required for effective use

This range includes:

- EMS 'transport' ventilators (this corresponds to the European definition of an emergency ventilator).
- Extended 'transport ventilators( corresponding to the European definition of a transport ventilator). These have more advanced alarm and monitoring function than the EMS ventilators
- Portable ventilators with an internal compressor
- Home ventilators

The authors noted the following about portable ventilators:

- The devices included in each subgroup are capable of ventilating all but the sickest of patients with ARF
- EMS 'transport' ventilators allow respiratory rate and tidal volume to be set but have limited versatility
- Devices having patient triggering capabilities and trigger mechanisms that compensate for device-provided PEEP are preferred
- Existing portable ventilators provide a broad range of alarms ranging from minimal to extensive.
- Devices without a disconnection and apnoea alarm will require constant operator attendance.
- Monitoring with alarms makes it possible for staff to be remote from the patient, thus reducing the risk of contagion
- Devices with an internal compressor and oxygen blender will be the most oxygen sparing.

4. ICU ventilators

The authors noted that full specification ventilators may be required for a proportion of patients but that the numbers cannot be predicted. Moreover the costs and difficulty of operating such ventilators make them unsuitable for mass procurement and stockpiling

(Author's note: At the time of writing, the COVID19 pandemic has shown that this is still the case and ICU ventilator availability has been stretched to the limit in some countries).



### **12.4.2 *France***

The emergency medical service in France (SAMU) is operated by anesthesiologists and emergency physicians. These are able to provide IPPV at the site of an emergency on a routine basis using mobile intensive care units. Over the years SAMU has used two control gas powered ventilators (Chap. 7) of the type used in hospital operating theatre units for transport between theatre and the recovery area and the ICU. These devices have the advantage that they are simple to operate and are familiar to the theatre and nursing staff who would be able to operate them under medical supervision in the event of mass ventilation. To address the problem of mass ventilation SAMU procured nearly 2000 two control gas-powered ventilators which are stockpiled around the country. This type of ventilator, allowing variation of the tidal volume and frequency with the ability to react to the patient's own respiratory efforts (using a CMV/demand system) together with the ability to provide PEEP offers a practical solution to the problem of mass ventilation in terms of equipment that is simple and understood by a large number of potential operators who can be used in HDU when normal ICU ventilation facilities are saturated.

More recently the SAMU mobile intensive care units have been equipped with electronically controlled turbine ventilators which can provide more adaptive ventilation than pneumatic ventilators. This has broadened the ventilation experience of many physicians and general practitioners who work with the service on a part time basis. Such experience will help to manage mass ventilation requirements in the future.

### **12.4.3 *United Kingdom***

Portable gas-powered ventilators are widely-used in the UK both by the ambulance services and in hospital EDs. During the first decade of the new century nearly a thousand Pneupac VR1 ventilators were procured by the UK Department of Health for use in chemical and other mass hazards requiring ventilation and the plan was to deploy these in the first instance for mass ventilation in the case of a pandemic. However, the stockpile was apparently not maintained and the COVID19 pandemic saw a renewed procurement of two control ventilators like the ParaPac Plus to provide an emergency reserve for ventilation of patients not yet requiring complex ICU support. There was also an initiative to produce a completely new ventilator using engineering companies with no previous experience in this field. This proved to be more difficult than thought and at the time of writing (June, 2020) mass production of the 'Rapidly Manufactured Ventilator System' has not started. The specifications behind the project are shown in Box 12.2 The very limited success of this Government project highlights the need to procure and importantly, to maintain stockpiles of ventilators before an event requiring mass ventilation occurs.

**Box 12.2 UK Government 2020 Guidelines for a Rapidly Manufactured Ventilator System (RMVS)**

1. Modes: must have at least 1, optionally 2 modes of ventilation as follows:
  - a. Must have CMV.
  - b. The CMV mode must be either
    - i. (ideally) Pressure Regulated Volume Control (PRVC), or
    - ii. pressure controlled ventilation (PCV), or
    - iii. minimally a volume controlled ventilation (VCV).
  - c. PRVC/Pressure Controlled—a set pressure is delivered for the period of inspiration and the volume achieved is measured and displayed. Ideally PRVC, an adaptive mode where the tidal volume is set and the lowest possible pressure is delivered to achieve this volume. PCV where the user has to provide the adaptive control to achieve tidal volume is only acceptable if the tidal volume delivered is clearly displayed and the user can set patient specific upper and lower tidal volume alarms to alert to the need to adjust the pressure.
  - d. Volume Control Ventilation—the user sets a tidal volume and respiratory rate. The tidal volume is delivered during the inspiratory period. Acceptable only if additional pressure limiting controls are available.
  - e. Should have a spontaneous breathing pressure support mode for those patients breathing to some extent themselves, e.g. BIPAP or SIMV-PC. The user sets an inspiratory pressure and an expiratory pressure. The ventilator can sense when a patient starts to breathe in and applies the inspiratory pressure, then detects when the patient starts to breathe out and apply the expiratory pressure (this pressure is still positive but lower than the inspiratory pressure).
2. If a pressure support mode is provided the RMVS must failsafe automatically onto mandatory ventilation if the patient stops breathing in this mode.
3. Inspiratory airway pressure, the higher pressure setting that is applied to make the patient breathe in;
  - a. Plateau pressure should be adjusted to achieve volume and must be limited to 35 cm H<sub>2</sub>O by default. It is acceptable if an option to increase this to 70 cm H<sub>2</sub>O in exceptional circumstances is provided. This must require a positive decision and action by the user.
  - b. Peak pressure should be no more than 2 cm H<sub>2</sub>O greater than plateau pressure.

- c. If volume control ventilation is used, the user must be able to set inspiratory airway pressure limit in the range at least 15–40 cm H<sub>2</sub>O in at least increments of 5 cm H<sub>2</sub>O.
- d. There must be a mechanical fail - safe valve that opens at 80 cm H<sub>2</sub>O.
- 4. Positive End Expiratory Pressure (PEEP). The pressure maintained in the breathing system during expiration.
  - a. RMVS must provide a range 5–20 cm H<sub>2</sub>O adjustable in 5 cm H<sub>2</sub>O increments.
  - b. PEEP must be maintained during expiration
  - a. RMVS must provide I:E of 1:2 as the default setting.
  - b. RMVS could provide adjustable I:E in the range 1:1–1:3.
  - a. RMVS must provide a range 10–30 breaths per minute in increments of 2 (only in mandatory mode) that can be set by the user.
    - a. Must have at least one setting of  $400 \pm 10$  ml.
    - b. Should have 350 ml and 450 ml options.
    - c. Could have a range 250–600 ml in steps of 50 ml.
    - d. Could have a range up to 800 ml.

### Monitoring and Alarms

IEC60601-1-8:2006 is the one relevant standard for alarms for RMVS. Alarms, alarm limits, and priorities are complex areas to optimise for human usability. The key is to get enough alarms but not too many and for alarms to be clearly ranked so that more urgent patient safety problems are highlighted more. Early attention to this area is important, and should be built in from the start.

1. Must alarm at:
  - a. Gas or electricity supply failure.
  - b. Machine switched off while in mandatory ventilation mode.
  - c. Inspiratory airway pressure exceeded.
  - d. Inspiratory and PEEP pressure not achieved (equivalent to disconnection alarm).
  - e. Tidal volume not achieved or exceeded.
2. Monitoring displayed continuously so the user can verify.
  - a. Must show the current settings of tidal volume, frequency, PEEP, FiO<sub>2</sub>, ventilation mode.
  - b. Must show the actual current airway pressure
  - c. Should show the achieved tidal volume, breathing rate, PEEP, and FiO<sub>2</sub>.

- d. If pressure support mode is provided, there must be real time confirmation of each patient breath and an alarm if below acceptable range.
- e. Could provide CO<sub>2</sub> monitoring.

### **Miscellaneous**

1. Must be reliable. RMVS must be capable of continuous operation (100% duty cycle) for 14 days.
2. The expected durability must be specified.
3. Could be floor standing.
4. Could be small and light enough to mount on patient bed with orientation independent functioning.
5. Should be as robust as possible. For example, it may be dropped from bed height to floor.
6. It must be intuitive to use for qualified medical personnel, but these may not be specialists in ventilator use.
  - a. Must not require more than 30 min training for a doctor with some experience of ventilator use.
  - b. Must include Instructions for Use.
  - c. Instructions for use should be built into the labelling of the ventilator, e.g. with 'connect this to wall' etc.
  - d. Must include clear labelling of all critical functions and controls using standard terms, pictograms and colours that will be readily recognised by UK healthcare staff.
7. Must have transparent design, supply chain, manufacture, quality assurance and testing processes that are of sufficient quality to enable MHRA officials to deem appropriate for use in exceptional circumstances.
8. Must not be excessively cumbersome so that it would impede hospital operations or prevent easy movement within hospital premises.
9. Must be made from materials and parts readily available in the UK supply chain (anticipating increasing global restrictions on freight movement).

One advantage of the UK approach in providing portable gas-powered ventilators for mass ventilation is that they are in everyday emergency use in the ambulance and hospital services and are familiar to a wide range of users who could be called upon to provide mass ventilation. This makes their use in an HDU easier.

The provision of ventilators is only one part of the problem in responding to the need for mass ventilation. There is also the problem of obtaining enough disposables like circuits and airways which are an essential part of IPPV. This proved to be difficult in a world market that was overloaded at the time. Another problem was the supply of medical oxygen which led to the consideration of using air as the driving gas for pneumatic ventilators supplemented by oxygen at atmospheric pressure which gives considerable efficiency in the use of the gas. This problem is not new.

**Fig. 12.1** Multiple Pneupac 2R resuscitation ventilators driven by a central air compressor for mass ventilation (Photograph courtesy of Pneupac Ventilation, Smiths Medical International, Luton, United Kingdom)



In the early 1990s the UK produced a system with multiple small ventilators with multiple small PGPV operated from a central compressed air supply (Fig. 12.1) for use in desert warfare conditions where mass ventilation was expected after chemical agent attacks

More recently, it has been shown that a pneumatic transport ventilator (Pneupac Ventipac) can be driven on compressed air and the oxygen concentration of the gas delivered to the patient can be supplement at the level of the patient valve using free flow oxygen from a cylinder or an oxygen concentrator (Burchell and Baker 2014). This concept is attractive as an option when compressed oxygen supplies may be scarce. To operate while being driven by compressed air rather than oxygen the ventilator controls require recalibrating against a test lung. The ventilator then delivers a recalibrated tidal volume and oxygen is injected on a free flow basis through a T piece sited at the end of the patient circuit before the patient valve. Box 12.3 shows this and also the delivered oxygen concentrations that can be delivered to the patient with an increasing flow of added oxygen. The second graph shows that this does not significantly affect the delivered tidal volume. This approach to the use of portable pneumatic ventilators may be of considerable value, not only in mass

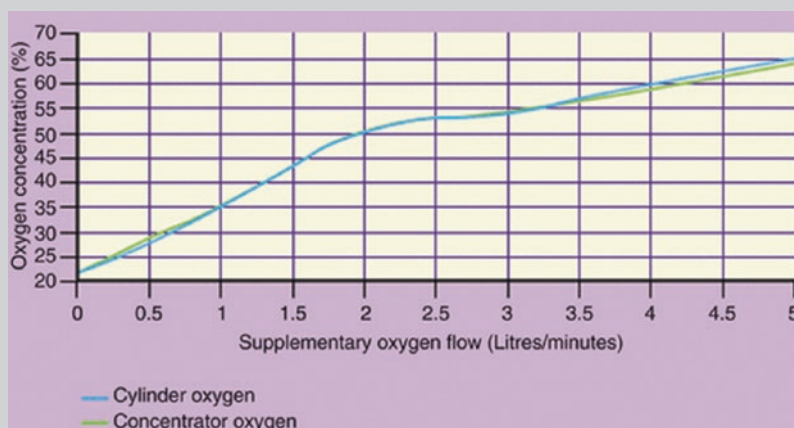
ventilation for a pandemic but also in parts of the world where oxygen supply is often variable.

Recognising the importance of the supply of oxygen during the COVID19 pandemic the WHO issued guidelines for the procurement and estimating of the amounts required in 2020. These are cited in the suggestions for further reading at the end of the chapter.

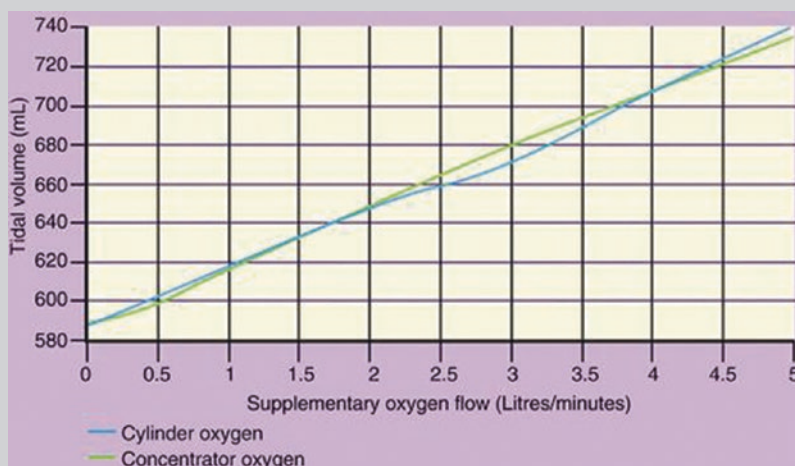
**Box 12.3 Enhancing the Delivered  $\text{FiO}_2$  from a Pneumatic Ventilator Driven by Compressed Oxygen by Adding Free Flow Oxygen to the Patient Circuit (Burchell and Baker 2014)**



(a) Modified VentiPac patient circuit with additional oxygen delivered by a T piece before the patient valve



(b) Increased delivered  $\text{FiO}_2$  with oxygen supplementation from a cylinder or oxygen concentrator supply



(c) Increase in delivered  $V_t$  with supplemental oxygen

## 12.5 Conclusions

1. Many countries around the world now recognise the requirement to provide mass artificial ventilation following respiratory epidemics and release of toxic agents.
2. The COVID19 pandemic has underlined the need for a mass ventilation capability together with sufficient stockpiles of ancillary disposable equipment such as patient circuits and oxygen to provide a timely and adequate response to the need for respiratory support.
3. The mass provision of sophisticated ICU ventilators to respond to expanded patient numbers has been shown to be difficult due to (a) very high costs (b) the lack of the skilled medical and nursing staff required to operate such machines.
4. A number of publications, mostly from the United States have examined the provision of mass ventilation in detail. These are described, along with planning and equipment provision in France and the United Kingdom. There is now a far greater awareness of the need for mass ventilation in the large number of countries around the world that have been affected by the 2020 pandemic.
5. Most countries are opting for mass procurement of simple gas powered ventilators which can provide basic support. These can then be supplemented in complicated cases by more complex machines in hospital and by electronic turbine transport ventilators which are more complex to operate but less expensive than ICU machines.
6. There is no one single solution that will suit all cases in a pandemic. A flexible solution will be required to match the number of cases requiring ventilation with the setting, the available equipment and staff who are able to operate it.

## Suggestions for Further Reading

- Anon. American Association for Respiratory Care. Guidelines for Acquisition of Ventilators to Meet Demands for Pandemic Flu and Mass Casualty Incidents. 2006 (at [info@aacrc.org](mailto:info@aacrc.org), accessed 13/11/15)
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## Chapter 13

# Conclusions



1. Since the establishment of intermittent positive pressure ventilation as a standard practice in the mid-twentieth century there has been an increasingly widening gap between specialised hospital and prehospital emergency ventilators. ICU ventilators have become highly sophisticated and computer-controlled, allowing ventilation that essentially supports the patient's own breathing efforts in an adaptive way. Portable ventilators have, however retained basic modes of ventilation designed to provide total or partial respiratory support.
2. Despite their simplicity of design and control, portable ventilators are often intimidating for the non-specialist provider of artificial ventilation who naturally feels that ventilation using a bag-valve device is easier to understand and safer.
3. Bag-valve ventilation is widely used in emergency practice but a better alternative is the portable ventilator. Such devices now cover a wide range, from simple pneumatic resuscitation ventilators through to complex electronically-controlled devices and the choice for the user is often bewildering.
4. The recognition of developing and complete respiratory failure is an essential step in preparing management by artificial ventilation. Here clinical skills remain essential but there are an increasing number of aids to monitoring such as pulse oximetry and capnography which can be used in non-hospital settings.
5. A number of studies have been published comparing the features of portable ventilators currently available on the market and these may be of some help to the user when deciding which ventilator to buy. The performance of many portable ventilators can be very variable in terms of calibration, particularly when being used with air mix and with clinical situations where lung compliance is reduced and airway resistance increased.
6. Bag valve devices have been used in CPR for many years since they are relatively simple to use can deliver multiple ventilations required as part of basic life support protocols, However, most resuscitation ventilators available on the market now have the ability to deliver manually-controlled breaths to link in with the

delivered chest compressions. The ratio recommended remains at 30:2 with delivered ventilations of not less than 1s.

7. Artificial ventilation has been one of the major successes of clinical medicine in recent times, will continue to be a key area of practice for many specialties.
8. Perhaps the biggest challenge for prehospital emergency ventilation over the next decade will be to produce artificial ventilation that is more adapted to the delicate structure and physiology of the lungs, following the precedent set in intensive care medicine more than a decade ago.

### **13.1 Artificial Ventilation: Past Present and Future**

1. The chapters in this book have presented the origins, basic principles and practice of artificial ventilation at a level which will hopefully be helpful to the non-specialist. This approach is important given the increasing complexity of modern mechanical ventilators. With the development of advanced life support, both for physical and toxic trauma and for resuscitation, artificial ventilation plays an increasingly important role in modern emergency medicine. An understanding of the advantages and potential problems of artificial ventilation is essential for modern emergency care and for the transport of ventilator dependent patients. In addition, hazards such as epidemics or toxic releases can cause mass casualties who will require artificial ventilation as part of life support. In such situations, the conventional ventilation facilities of hospital ICU will almost certainly be overwhelmed and there will be a requirement for high dependency units manned by medical and nursing personnel who are comfortable with the use of portable ventilators, which although far simpler in their design and operation are able to deliver essential ventilation to maintain life in most cases of respiratory failure.
2. The history of artificial ventilation over the past three centuries has moved between negative pressure ventilation, copying the natural process of breathing and positive pressure ventilation which is essentially non-physiological but is effective and can be used outside the hospital. Intermittent positive pressure ventilation is now firmly established for the management of both total and partial respiratory failure. From its early beginnings in the polio epidemics of the 1950s there has been a rapid development of both hospital and pre-hospital positive pressure artificial ventilation. In addition, there has been an increasingly widening gap between the two practices with ICU ventilators becoming highly sophisticated and computer-controlled, allowing ventilation that essentially supports the patient's own breathing efforts in a highly adaptive way. Portable ventilators have however, retained basic modes of ventilation designed to provide total or partial respiratory support. But these too are becoming more complex and there is an overlap between portable ventilators and the ICU models. This has been highlighted by the development in recent years of increasingly compact computer-controlled ventilators that provide essentially the often

complex supportive ventilation used in the ICU for the management of ARDS and other severe respiratory conditions.

3. Despite their simplicity of design and control, portable ventilators are often intimidating for the non-specialist provider of artificial ventilation who naturally feels that ventilation using a bag-valve device is easier to understand and safer. From the author's experience it is a common comment among paramedical personnel that ventilation training does not receive nearly as much attention as other areas of practice such as advanced cardiac and trauma life support. The paucity of articles concerning emergency ventilation which provides the basis for the ILCOR resuscitation guidelines supports this view.
4. Better awareness and training for artificial ventilation should be a priority for emergency and other services. An understanding of the basic anatomy and physiology is a valuable start point and many ambulance services include these as part of the basic training curriculum. Armed with an understanding of the normal processes of breathing and respiration, the ventilation provider will then be better able to understand the pathophysiology of respiratory failure and the standard conditions which cause it. Chronic and acute infections are seen in everyday practice, with chronic obstructive airways disease and asthma being perhaps the most familiar. Equally important however is toxically-induced respiratory failure, both from self-poisoning and use of drugs of abuse and accidental or deliberate exposure to toxic chemicals which affect the respiratory system.
5. The recognition of developing and complete respiratory failure is a further essential step in preparing management by artificial ventilation. Here, clinical skills remain essential but there are an increasing number of aids to monitoring such as pulse oximetry and capnography which can be used in non-hospital settings. The history of the presenting condition and previous history provide a good basis for determining whether the respiratory failure can be classed as type 1 or type 2 and for planning appropriate treatment.
6. Providing treatment for respiratory failure covers a spectrum of potential techniques ranging from provision of free-flow oxygen to a patient who is still breathing to complete respiratory support for total respiratory failure. This spectrum covers a range of options such as CPAP and PEEP in addition to the modes usually found on portable ventilators. The use of the bag-valve device remains basic to many emergency services. While there has always been recognition of the dangers of gastric insufflation when using the BVM with an unprotected airway there is now increasing awareness of the dangers of barotrauma and volutrauma.
7. The alternative to the use of bag-valve ventilation in emergency is the portable ventilator and the function and operation of these has been considered in detail earlier. Such devices now cover a wide range, from simple pneumatic resuscitation ventilators through to complex electronically-controlled devices and the choice for the user is often bewildering. A number of studies have been published comparing the features of portable ventilators currently available on the market and these may be of some help to the user when deciding which ventila-

tor to buy. Information from manufacturers may also be of some help but should be treated with caution as the performance of many portable ventilators can be very variable in terms of calibration, particularly when being used with air mix and with clinical situations where lung compliance is reduced and airway resistance increased. Pressure - generating portable ventilators are particularly vulnerable in such situations and the flow generators which deliver a preset tidal volume are a better option.

8. Although flow generators have pressure limitation devices, the potential for barotrauma and volutrauma remains. These have been successfully addressed in the ICU with protective ventilation such as lower tidal volumes with permissive hypercapnia but the potential harm that can be caused by overventilation in emergency is beginning to be understood in the prehospital setting and should lead to more controlled and adaptive emergency and transport ventilation in the future. In particular, there may be a new approach to adaptive pressure support ventilation in emergency without the risk of inadequate delivery of tidal volume which has been a problem with pressure support modes in the past.
9. There will be a number of factors that go into the selection and use of a portable ventilator for any particular service including training skills and cost. It is essential that whatever device is chosen it should be thoroughly tested, preferably against a test lung before being used clinically. Training covering all aspects of the care and use of portable ventilators, preferably using clinical simulators is essential before using the ventilator on a patient. Complete familiarity with the operation and controls of any chosen ventilator is essential.
10. Management and monitoring of patients being ventilated is essential both for emergency and transport ventilation. Transport of ventilator-dependent patients is increasingly important both inside the hospital and from one hospital to another, sometimes over a distance of several thousand miles. This also highlights the importance of knowing how any particular ventilator will operate in conditions of high and low pressure. Ventilation under extreme circumstances where technical support and essentials such as compressed gases may be limited is also an important consideration. While paediatric ventilation is essentially the area of the specialist, it is important for other medical and nursing staff to understand the differences between this and adult artificial ventilation and the potential hazards. Many adult portable ventilators can be used to ventilate children down to 10 or 5 Kg in weight and this is valuable when no specific paediatric ventilator is available.
11. Another area of interest is the requirement for positive pressure ventilation during CPR. The ILCOR guidelines of 2010 heralded the idea of chest compressions without rescue breathing, based upon surveys that had indicated public reluctance to use mouth to mouth breathing. Consequently, the 2015 guidelines have confirmed that chest compression without ventilation is acceptable if no method of artificial ventilation is available, or bystanders are unwilling to provide it. The value of the bag-valve mask in primary CPR is recognised and most resuscitation ventilators available on the market now have the ability to deliver manually-controlled breaths to link in with the delivered chest compressions.

The ratio recommended remains at 30:2 with delivered ventilations of not less than 1s. The disadvantage of zero ventilation CPR is the oxygen debt that is built up after a few minutes and which must be overcome once return to spontaneous circulation is achieved. It is to be hoped that a wider use of resuscitation ventilators in primary CPR in the future will overcome this problem and lead to a better long term survival rate from primary cardiac arrest. The place of ventilation in preventing secondary cardiac arrest from respiratory failure is well-secured in this form of cardiac arrest which is common in children and as a sequel of toxic trauma. There remains a need however for more studies on emergency ventilation using portable ventilators in a clinical setting. However, until there is greater acceptance of the use of such devices by the emergency services around the world the number of studies is likely to be few, leading to a limited evidence data base.

12. Looking towards the future, it is likely that mechanical ventilation for both emergency and transport will continue to be a major part of prehospital practice, including long-distance transport for medical evacuations with the continuing rise of tourism. Another factor leading to an increase in more local transport ventilation is the increasing specialisation of hospital facilities with the creation of regional neuro- and cardiac surgical centres. In response to the threats (and with COVID 19, the reality) of epidemics and of terrorist attacks in the urban setting, smaller regional hospitals will require high dependency units equipped with basic ventilators that can be used effectively by non-specialists, to liberate ICU beds for the worst-affected cases.
13. While highly specialised ventilators will continue to become more complex and reliant on computer technology, there will still be a need for basic ventilation and devices that can be operated by non-specialists. Complexity is already beginning to move into the prehospital area but many of the potential operators do not understand the complex controls and are not able to deploy the devices quickly. Simple gas-powered ventilators are likely to continue and will be developed without being over complicated. Monitoring and safety devices will continue to be important, as a backup to careful clinical practice. Portable devices are likely to become more interactive with the adoption of modes such as pressure support increasingly available on sophisticated transport ventilators.
14. Perhaps the biggest challenge for prehospital emergency ventilation over the next decade will be to produce artificial ventilation that is more adapted to the delicate structure and physiology of the lungs. It was realised in the hospital ICU world over a decade ago that over-ventilation with the use of high tidal volumes and inflation pressures was damaging the lungs, leading to ARDS. The adoption of a completely different approach to ventilation in the ICU with far smaller tidal volumes and a structured use of PEEP (the 'Open Lung' strategy) has led to a great improvement in the management of ARDS. It is possible that, in cases of severe trauma excessive artificial ventilation in the emergency phase, as is often the case using BVM devices may be leading to the development of later ARDS in the hospital. The lesson may be learned from the hospital studies

of the last 15 years but there will be a need for careful prospective clinical studies to confirm and correct ventilation practice in pre-hospital practice. The COVID 19 pandemic has led to intensive review of ventilation strategies within the ICU and hopefully will fuel more studies in artificial ventilation in other areas.

15. In any event artificial ventilation, one of the successes of clinical medicine in recent times, will continue to be a key area of practice for many years to come and particularly requires further study in the effective use of portable ventilators and a wider understanding of more complex ventilation modes used in the hospital. There is an increasing pace of technological developments in the field. It is important that ventilation providers should understand these and use the devices available to best effect with appropriate clinical skills.

# Appendix A: The 2015 ILCOR Resuscitation Guidelines: An Analysis of the Implications of Recommendations for Ventilation Management

## A.1 Introduction

Guidelines for cardiopulmonary resuscitation (CPR) are published every 5 years by the International Liaison Committee on Resuscitation (ILCOR). The committee includes representatives from the American Heart Association (AHA), the European Resuscitation Council (ERC), the Heart and Stroke Foundation of Canada (HSFC), the Australian and New Zealand Committee on Resuscitation (ANZCOR), the Resuscitation Council of Southern Africa (RCSA), the Inter-American Heart Foundation (IAHF), and the Resuscitation Council of Asia (RCA).

Since 2000, researchers from the ILCOR member councils have evaluated resuscitation science in 5-yearly cycles. The most recent International Consensus Conference was held in Dallas in February 2015 and the published conclusions and recommendations from this process form the basis of the ILCOR Guidelines which were published simultaneously in the journals *Circulation* and *Resuscitation*. The next review of the guidelines is due late in 2020 following conferences to be held by the European Resuscitation Council and the American Heart Association. At the time of preparing this edition it is not clear whether these meetings will go ahead. Consequently the 2015 guidelines cited here should still be regarded as valid. However the reader is advised to check any later modifications which are normally published in the journals *Resuscitation* and *Circulation*.

The guidelines are constructed from systematic reviews of all scientific and clinical papers concerning all aspects of CPR which had been published during the previous 5 years. The reviewing process is carried out by international experts working according to strict guidelines about how the evidence is assessed and the exact language in the way the guidelines are expressed. The basis of the review exercise is to base all CPR upon sound scientific evidence. There are many components to cardiopulmonary resuscitation and not all these are reviewed during each 5 year cycle.

There is always great interest in the publication of new ILCOR guidelines from all those who may be involved in CPR. These include paramedical and medical

personnel as well as public first responder training, fire, police and other non-medical services. ILCOR emphasises that the published guidelines are not necessarily binding. This means that they can be open to interpretation by medically-trained personnel who can make their own clinical decisions according to the circumstances. However, for non-medical personnel the guidelines are regarded as rules which are followed rigidly in training programmes.

The 2015 ERC Guidelines, published in the journal *Resuscitation* state specifically that ‘they do not define the only way that resuscitation can be delivered; they merely represent a widely accepted view of how resuscitation should be undertaken both safely and effectively. The publication of new and revised treatment recommendations does not imply that current clinical care is either unsafe or ineffective.’

## **A.2 Publication of the Guidelines**

Up to 2010 the published guidelines in *Resuscitation* and *Circulation* were almost identical but this has not been the case with the latest guidelines which were published in October 2015. The AHA guidelines are published in 15 parts which cover all aspects of adult and paediatric CPR. They are set out with the reference base and evidence ratings as before but the 2015 guidelines included a Master List of Recommendations which cover the year when the topic was last reviewed and current recommended advice. Not every topic relating to CPR is reviewed every 5 years and this table helps to clarify this point and emphasize that the recommendations from the previous guidelines are still valid, even if they have not been reviewed again as part of the current guidelines.

The ERC published guidelines do not make this clear, nor do they publish specific levels of evidence for recommendations as does the AHA. Consequently, for a topic like the use of portable automatic ventilators in CPR and post CPR care, the 2015 AHA guidelines give a clear recommendation for their use while the subject receives almost no attention in the ERC guidelines. This is markedly different from their 2010 guidelines which discussed ventilators in some detail. The AHA guidelines also make it clear that ventilation has not been reviewed in the past 5 years and that the 2010 guidelines are still valid.

## **A.3 The Basis of the Evidence**

Assessment of ventilation in CPR from an evidence based-standpoint is made difficult by the relatively small numbers of published studies on the topic compared with many papers relating to the restoration and management of circulation and the management of airways. Thus recommendations for ventilation are based on a limited number of publications and therefore do not usually have a high evidence rating. Nevertheless, the 2015 ILCOR guidelines do contain clear recommendations about ventilation based on what evidence is available and this paper summarises the current guidelines and their application, together with new recommendations for monitoring which are of importance to the use of ventilation devices.



### A.3.1 *Presentation of Evidence Levels and the Wording of the Guidelines*

The 2015 AHA guidelines present the way that scientific evidence relating to resuscitation is analysed and the form of wording that should be used for recommendations. A synopsis of this process is shown below. It will be seen in the guidelines that the words ‘it is reasonable to ...’ appear frequently in relation to artificial ventilation. This choice of wording is highly arbitrary and, in my opinion inappropriate for a subject that is embedded in modern medicine and has obvious proven value. To use the words ‘it is reasonable to use artificial ventilation ...’ for the management of a child that has stopped breathing where artificial ventilation is essential and life-saving highlights the problem of the subjective system of wording chosen by AHA. The established life saving position of artificial ventilation, set out in the various chapters of this book show that it should be described as ‘highly desirable’ at the very least. The rationale of the AHA statements of evidence can be found at [https://www.ahajournals.org/toc/circ/132/18\\_suppl\\_2](https://www.ahajournals.org/toc/circ/132/18_suppl_2)

CLASS (STRENGTH) OF RECOMMENDATION	LEVEL (QUALITY) OF EVIDENCE‡
<b>CLASS I (STRONG)</b> Benefit >>> Risk Suggested phrases for writing recommendations: <ul style="list-style-type: none"> <li>■ Is recommended</li> <li>■ Is indicated/useful/effective/beneficial</li> <li>■ Should be performed/administered/other</li> <li>■ Comparative-Effectiveness Phrases†:             <ul style="list-style-type: none"> <li>○ Treatment/strategy A is recommended/indicated in preference to treatment B</li> <li>○ Treatment A should be chosen over treatment B</li> </ul> </li> </ul>	<b>LEVEL A</b> <ul style="list-style-type: none"> <li>■ High-quality evidence‡ from more than 1 RCTs</li> <li>■ Meta-analyses of high-quality RCTs</li> <li>■ One or more RCTs corroborated by high-quality registry studies</li> </ul>
	<b>LEVEL B-R (Randomized)</b> <ul style="list-style-type: none"> <li>■ Moderate-quality evidence‡ from 1 or more RCTs</li> <li>■ Meta-analyses of moderate-quality RCTs</li> </ul>
<b>CLASS IIa (STRONG)</b> Benefit >>> Risk Suggested phrases for writing recommendations: <ul style="list-style-type: none"> <li>■ Is reasonable</li> <li>■ Can be useful/effective/beneficial</li> <li>■ Comparative-Effectiveness Phrases†:             <ul style="list-style-type: none"> <li>○ Treatment/strategy A is probably recommended/indicated in preference to treatment B</li> <li>○ It is reasonable to choose treatment A over treatment B</li> </ul> </li> </ul>	<b>LEVEL B-NR (Nonrandomized)</b> <ul style="list-style-type: none"> <li>■ Moderate-quality evidence‡ from 1 or more well-designed, well-executed nonrandomized studies, observational studies, or registry studies</li> <li>■ Meta-analyses of such studies</li> </ul>
	<b>LEVEL C-LD (Limited Data)</b> <ul style="list-style-type: none"> <li>■ Randomized or noneandonized observation or registry studies with limitations of design or execution</li> <li>■ Meta-analyses of such studies</li> <li>■ Physiological or mechanistic studies in human subjects</li> </ul>
<b>CLASS IIb (WEAK)</b> Benefit ≥ Risk Suggested phrases for writing recommendations: <ul style="list-style-type: none"> <li>■ May/might be reasonable</li> <li>■ May/might be considered</li> <li>■ Usefulness/effectiveness is unknown/unclear/uncertain or not well established</li> </ul>	<b>LEVEL C-EO (Expert Opinion)</b> <ul style="list-style-type: none"> <li>■ Consensus of expert opinion based on clinical experience</li> </ul>
<b>CLASS III: No Benefit (MODERATE)</b> Benefit = Risk (Generally, LOE A or B use only) Suggested phrases for writing recommendations: <ul style="list-style-type: none"> <li>■ Is not recommended</li> <li>■ Is not indicated/useful/effective/beneficial</li> <li>■ Should not be performed/administered/other</li> </ul>	COR and LOE are determined independently (any COR may be paired with any LOE). A recommendation with LOE C does not imply that the recommendation is weak. Many important clinical questions addressed in guidelines do not lend themselves to clinical trials. Although RCTs are unavailable, there may be a very clear clinical consensus that a particular test or therapy is useful or effective. * The outcome or result of the intervention should be specified (an improved clinical outcome or increased diagnostic accuracy or incremental prognostic information).
<b>CLASS III: Harm (STRONG)</b> Risk > Benefit Suggested phrases for writing recommendations: <ul style="list-style-type: none"> <li>■ Potentially harmful</li> <li>■ Causes harm</li> <li>■ Associated with excess morbidity/mortality</li> <li>■ Should not be performed/administered/other</li> </ul>	† For comparative-effectiveness recommendations (COR I and IIa; LOE A and B only), studies that support the use of the comparator verbs should involve comparisons of the treatments or strategies being evaluated. ‡ The method of assing quality is evolving, including the application of standardized, widely used, and preferably validated evidence grading tools; and for systematic reviews, the incorporation of an Evidence Review Committee. COR indicates Class of Recommendation: EO, expert opinion; LD, limited data; LOE, Level of Evidence: NR, nonrandomized; R, randomized; and RCT, randomized controlled trial.

## **A.4 ERC and AHA 2015: The Main Changes from the 2010 Guidelines for Airway and Ventilation Management**

Overall, there have been no substantial changes to fundamental CPR with the chest compression to ventilation ratio remaining at 30:2 for basic life support with an unprotected airway and 100 compressions per minute with 10 ventilations per minute for protected airway. For rescue and bag valve ventilation the inspiratory time remains at 1s.

### ***A.4.1 Basic Adult Life Support***

The ERC version of the guidelines lists the following key changes from the 2010 guidelines:

- (i) CPR providers should perform chest compressions for all victims in cardiac arrest. CPR providers trained and able to perform rescue breaths should combine chest compressions and rescue breaths. Our confidence in the equivalence between chest compression-only and standard CPR is not sufficient to change current practice.
- (ii) High-quality CPR remains essential to improving outcomes. The guidelines on compression depth and rate have not changed. CPR providers should ensure chest compressions of adequate depth (at least 5 cm but no more than 6 cm) with a rate of 100–120 compressions/min. After each compression allow the chest to recoil completely and minimise interruptions in compressions. When providing rescue breaths/ventilations spend approximately 1s inflating the chest with sufficient volume to ensure the chest rises visibly. The ratio of chest compressions to ventilations remains at 30:2.
- (iii) Do not interrupt chest compressions for more than 10 s to provide ventilations.

The AHA guidelines list the following key changes from the 2010 BLS guidelines

1. More data are available indicating that high-quality CPR improves survival from cardiac arrest.

Components of high-quality CPR include:

- Ensuring chest compressions of adequate rate
- Ensuring chest compressions of adequate depth
- Allowing full chest recoil between compressions
- Minimizing interruptions in chest compressions
- Avoiding excessive ventilation

Recommendations are made for a simultaneous, choreographed approach to performance of chest compressions, airway management, rescue breathing, rhythm

detection, and shock delivery (if indicated) by an integrated team of highly trained rescuers in applicable settings.

2. It is reasonable for healthcare providers to provide chest compressions and ventilation for all adult patients in cardiac arrest, from either a cardiac or a noncardiac cause (Class IIb, LOE C-LD).
3. When the victim has an advanced airway in place during CPR, rescuers no longer deliver cycles of 30 compressions and 2 breaths (i.e., they no longer interrupt compressions to deliver 2 breaths). Instead, it may be reasonable for the provider to deliver 1 breath every 6 s (10 breaths/min) while continuous chest compressions are being performed (Class IIb, LOE C-LD).
4. When the victim has an advanced airway in place during CPR, it may be reasonable for the provider to deliver 1 breath every 6 s (10 breaths/min) while continuous chest compressions are being performed (Class IIb, LOE C-LD). This simple rate, rather than a range of breaths per minute, should be easier to learn, remember, and perform.

### ***A.4.2 Adult Advanced Life Support***

The evidence base for airway and ventilation management during cardiopulmonary resuscitation remains very limited. The following statement in the executive summary of the ERC version of the ILCOR guidelines summarizes the situation:

‘The optimal strategy for managing the airway has yet to be determined. Several observational studies have challenged the premise that advanced airway interventions (tracheal intubation or supraglottic airways) improve outcomes. The ILCOR ALS Task Force has suggested using either an advanced airway (tracheal intubation or supraglottic airway (SGA) or a bag-mask for airway management during CPR. This very broad recommendation is made because of the total absence of high quality data to indicate which airway strategy is best. In practice a combination of airway techniques will be used stepwise during a resuscitation attempt. The best airway, or combination of airway techniques will vary according to patient factors, the phase of the resuscitation attempt (during CPR, after ROSC), and the skills of rescuers. A stepwise approach to airway and ventilation management using a combination of techniques is therefore suggested’.

AHA key changes for airway—ventilation

Significant New and Updated Recommendations: 2015

1. Based on new data, the recommendation for use of the maximal feasible inspired oxygen during CPR was strengthened. This recommendation applies only while CPR is ongoing and does not apply to care after return of spontaneous circulation (ROSC).
2. Continuous waveform capnography remained a Class I recommendation for confirming placement of an endotracheal tube. Ultrasound was added as an additional method for confirmation of endotracheal tube placement.

3. The AHA table of master recommendations for the use of portable ventilators during and after CPR states the following (based upon the analysis to 2010)

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1. Automatic Transport Ventilators

‘In both out-of-hospital and in-hospital settings, automatic transport ventilators (ATVs) can be useful for ventilation of adult patients in non-cardiac arrest who have an advanced airway in place’ (Class IIb, LOE C). (not reviewed in 2015)

2. Automatic Transport Ventilators

‘During prolonged resuscitative efforts the use of an ATV (pneumatically powered and time- or pressure-cycled) may allow the EMS team to perform other tasks while providing adequate ventilation and oxygenation’ (Class IIb, LOE C). (not reviewed in 2015)

AHA statements from the 2010 guidelines

- ‘ATV can be useful in an out-of-hospital setting in patients with an airway in place and who are not in cardiac arrest’
- ‘Very few studies have been published which evaluate the use of ATV during resuscitation’
- ‘During prolonged resuscitation, the use of ATV (gas-powered, time or pressure cycled) may allow EMS teams to perform other tasks while providing adequate ventilation and oxygenation’

ERC guidance on adult ALS ventilation

The ERC 2015 guidelines for adult ALS do not make any mention of portable ventilators. From the information provided by the AHA guidelines this may be explained by the fact that the topic has not been reviewed since 2010. However, unlike the AHA the ERC have not clarified this point.

The following statements concerning airway and ventilation management appear in the ERC version of the 2015 ILCOR guidelines:

Advanced Life Support providers should give artificial ventilation as soon as possible for any patient in whom spontaneous ventilation is inadequate or absent. Expired air ventilation (rescue breathing) is effective, but the rescuer’s expired oxygen concentration is only 16–17%, so it must be replaced as soon as possible by ventilation with oxygen-enriched air. The pocket resuscitation mask is similar to an anaesthetic facemask, and enables mouth-to-mask ventilation. It has a unidirectional valve, which directs the patient’s expired air away from the rescuer. The mask is transparent so that vomit or blood from the patient can be seen. Some masks have a connector for the addition of oxygen. When using masks without a connector, supplemental oxygen can be given by placing the tubing underneath one side and ensuring an adequate seal. Use a two-hand technique to maximise the seal with the patient’s face. High airway pressures can be generated if the tidal volume or

inspiratory flow is excessive, predisposing to gastric inflation and subsequent risk of regurgitation and pulmonary aspiration.

The risk of gastric inflation is increased by:

- of the head and neck, and an obstructed airway;
- incompetent oesophageal sphincter (present in all patients with cardiac arrest);
- high airway inflation pressure.

Conversely, if inspiratory flow is too low, inspiratory time will be prolonged and the time available to give chest compressions is reduced. Deliver each breath over approximately 1 s, giving a volume that corresponds to normal chest movement; this represents a compromise between giving an adequate volume, minimizing the risk of gastric inflation, and allowing adequate time for chest compressions. During CPR with an unprotected airway, give two ventilations after each sequence of 30 chest compressions. Inadvertent hyperventilation during CPR is common. While this increased intrathoracic pressure and peak airway pressures in a small case series in humans, a carefully controlled animal experiment revealed no adverse effects. We suggest a ventilation rate of 10 per minute during continuous chest compressions with an advanced airway based on very limited evidence.

**Self-inflating bag.** The self-inflating bag can be connected to a facemask, tracheal tube or supraglottic airway (SGA). Without supplementary oxygen, the self-inflating bag ventilates the patient's lungs with ambient air (21% oxygen). The delivered oxygen concentration can be increased to about 85% by using a reservoir system and attaching oxygen at a flow 10 l/min. Although a bag-mask enables ventilation with high concentrations of oxygen, its use by a single person requires considerable skill. When used with a face mask, it is often difficult to achieve a gas-tight seal between the mask and the patient's face, and to maintain a patent airway with one hand while squeezing the bag with the other. Any significant leak will cause hypoventilation and, if the airway is not patent, gas may be forced into the stomach. This will reduce ventilation further and greatly increase the risk of regurgitation and aspiration. The two-person technique for bag-mask ventilation is preferable. Several recent observational studies and a meta-analysis have documented better outcomes with use of bag-mask ventilation compared with more advanced airways (SGA or tracheal tube). However, these observation studies are subject to significant bias caused by confounders such as advanced airways not being required in those patients who achieve ROSC and awaken early. Once a tracheal tube or a SGA has been inserted, ventilate the lungs at a rate of 10 breaths/min and continue chest compressions without pausing during ventilations. The laryngeal seal achieved with a supraglottic airway (SGA) may not be good enough to prevent at least some gas leaking when inspiration coincides with chest compressions. Moderate gas leakage is acceptable, particularly as most of this gas will pass up through the patient's mouth.

**Waveform capnography during advanced life support (a new topic) in the 2015 ERC and AHA guidelines**

Waveform capnography enables continuous real-time EtCO<sub>2</sub> to be monitored during CPR. During CPR, EtCO<sub>2</sub> values are low, reflecting the low cardiac output generated by chest compression. There is currently no evidence that use of

waveform capnography during CPR improves patient outcomes, although the prevention of unrecognised oesophageal intubation is clearly beneficial. The role of waveform capnography during CPR includes:

- tracheal tube placement in the trachea.
- ventilation rate during CPR and avoiding hyperventilation.
- the quality of chest compressions during CPR. EtCO<sub>2</sub> values are associated with compression depth and ventilation rate and a greater depth of chest compression will increase the value. Whether this can be used to guide care and improve outcome requires further study.
- ROSC during CPR. An increase in EtCO<sub>2</sub> during CPR may indicate ROSC and prevent unnecessary and potentially harmful dosing of adrenaline in a patient with ROSC. If ROSC is suspected during CPR withhold adrenaline. Give adrenaline if cardiac arrest is confirmed at the next rhythm check.
- during CPR. Lower ETCO<sub>2</sub> values may indicate a poor prognosis and less chance of ROSC; however, we recommend that a specific EtCO<sub>2</sub> value at any time during CPR should not be used alone to stop CPR efforts. End-tidal CO<sub>2</sub> values should be considered only as part of a multi-modal approach to decision-making for prognostication during CPR.

ERC 2010 guidelines for the use of portable ventilators

In 2010 the ERC made the following comments about the use of portable ventilators in CPR which are still relevant in 2015 (See Deakin et al., Resuscitation 2010;81:1305–1352).

#### 1. Emphasis on avoiding high airway pressure (p1318)

‘The difficulty of using the self-inflating bag by one person is acknowledged. When using a bag valve device a two person technique is preferable.’

#### 2. Automatic ventilators

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‘Very few studies have addressed specific aspects of ventilation during advanced life support. Some data indicate that ventilation rates during CPR are excessive, although other studies have shown more normal ventilation rates’

‘An automatic ventilator or resuscitators provide a constant flow of gas to the patient during inspiration; the tidal volume delivered is dependent on the inspiratory time (a longer time provides a greater tidal volume). Because pressure in the airway rises during inspiration, these devices are often pressure limited to protect the lungs against barotrauma. An automatic ventilator can be used with either a facemask or other airway device (e.g. tracheal tube or supraglottic airway device)’

‘Automatic ventilators should be set initially to deliver a tidal volume of 6–7 ml/kg at 10 breaths per minute. Some ventilators have co-ordinated colour markings on the controls to facilitate easy and rapid adjustments for patients of different sizes .....and others are capable of sophisticated variations in respiratory parameters’

‘Automatic ventilators provide many advantages over alternative methods of ventilation. These are:

- i. In un-intubated patients the rescuer has both hands free for mask and airway alignment.
- ii. Cricoid pressure can be applied with one hand while the other seals the mask on the face
- iii. In intubated patients they free the rescuer for other tasks
- iv. Once set, automatic ventilators provide constant tidal volume, respiratory rate and minute volume. Thus they may help to avoid excessive ventilation.
- v. Automatic ventilators are associated with lower peak pressure than manual ventilation which reduces intrathoracic pressure, facilitates improved venous return to the heart and subsequent cardiac output.
- vi. Mannikin studies have shown a significant decrease in gastric insufflations when using manually-triggered, flow-limited, oxygen-powered resuscitators compared with a bag-valve mask devices. However human studies have not been done and no data are available’

## **A.5 Paediatric Basic and Advanced Life Support**

### ***A.5.1 AHA Paediatric Guidelines***

‘The asphyxial nature of the majority of pediatric cardiac arrests necessitates ventilation as part of effective CPR, and 2 large database studies documented worse 30-day outcomes with compression-only CPR compared with conventional CPR. For this reason, conventional CPR (chest compressions and rescue breaths) is a Class I recommendation (LOE B-NR) for children. However, because compression-only CPR is effective in patients with a primary cardiac event, if rescuers are unwilling or unable to deliver breaths, we recommend rescuers perform compression-only CPR for infants and children in cardiac arrest (Class I, LOE B-NR). Conventional CPR (chest compressions and rescue breaths) is a Class I recommendation (LOE B-NR)’.

### ***A.5.2 ERC Paediatric Guidelines***

Healthcare providers commonly provide excessive ventilation during CPR and this may be harmful. A simple guide to deliver an appropriate tidal volume is to achieve normal chest wall rise. Use a ratio of 15 chest compressions to 2 ventilations and a

compression rate of 100–120 per minute. Once the airway is protected by tracheal intubation, continue positive pressure ventilation at 10 breaths/min without interrupting the chest compressions. Take care to ensure that lung inflation is adequate during chest compressions. Once ROSC has been achieved, provide normal ventilation (rate/volume) based on the child's age, and by monitoring end-tidal CO<sub>2</sub> and blood gas values, to achieve a normal arterial carbon dioxide tension (PaCO<sub>2</sub>) and arterial oxygen levels. Both hypocarbia and hypercarbia are associated with poor outcomes following cardiac arrest. This means that the child with ROSC should usually be ventilated at 12–24 breaths/min, according to their age normal values.

'Bag mask ventilation (BMV) is effective and safe for a child requiring assisted ventilation for a short period. Assess the effectiveness of BMV by observing adequate chest rise, monitoring heart rate and auscultating for breath sounds, and measuring SpO<sub>2</sub>. Any healthcare provider with a responsibility for treating children must be able to deliver BMV effectively'.

## A.6 Monitoring of Breathing and Ventilation

Monitoring end-tidal CO<sub>2</sub> (EtCO<sub>2</sub>) with a colorimetric detector or capnometer confirms tracheal tube placement in the child weighing more than 2 kg, and may be used in pre-and in-hospital settings, as well as during any transportation of a child. A colour change or the presence of a capnographic waveform for more than four ventilated breaths indicates that the tube is in the tracheobronchial tree both in the presence of a per-fusing rhythm and during cardiopulmonary arrest. The absence of exhaled CO<sub>2</sub> during cardiopulmonary arrest does not guarantee tube misplacement since a low or absent EtCO<sub>2</sub> may reflect low or absent pulmonary blood flow. Although an EtCO<sub>2</sub> higher than 2 kPa (15 mmHg) may be an indicator of adequate resuscitation, current evidence does not support the use of a threshold EtCO<sub>2</sub> value as an indicator for the quality of CPR or for the discontinuation of resuscitation.

### Peripheral pulse oximetry

Clinical evaluation to determine the degree of oxygenation in a child is unreliable; therefore, monitor the child's peripheral oxygen saturation continuously by pulse oximetry. Pulse oximetry can be unreliable under certain conditions, e.g. if the child is in circulatory failure.

## A.7 Conclusions

1. This appendix has presented an analysis of the key points of the 2015 ILCOR guidelines concerning artificial ventilation. There have been no substantial changes from the 2010 guidelines (which are still valid) with the exception of the emphasis on waveform capnography. The interested reader is referred to the



original publications of the guidelines listed in the suggestions for further reading listed below.

2. Artificial ventilation remains a neglected area of study in cardiopulmonary resuscitation and this is reflected by the relatively small number of papers that appear each year on the subject. ILCOR notes that the database remains limited and has not conducted a review of artificial ventilation since 2010.

The AHA table of master recommendations is a valuable quick reference for new and existing guidelines.

### **Suggestions for Further Reading**

- Deakin CD, Nolan JP, Soar J, et al. European Resuscitation Council Guidelines for Resuscitation 2010, Section 4: Adult advanced life support. *Resuscitation*. 2010;81:1305–52.
- Koster RW, Baubin MA, Bossaert LL, et al. European Resuscitation Council Guidelines for Resuscitation 2010: Section 2. Adult basic life support and use of automated external defibrillators. *Resuscitation*. 2010;81:1277–92.
- Monsieurs KG, Nolan JP, Bossaert LL, et al. European Resuscitation Guidelines for Resuscitation 2015. Section 1: Executive summary. *Resuscitation*. 2015;95:1–80.
- Naimar RW, Shuster M, Calloway CW, et al. Part 1. Executive Summary. 2015 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2015;132(Suppl 2):S315–D367.

The full ERC and AHA 2015 guidelines are available for free downloading on the following internet websites:

<https://doi.org/10.1016/j.resuscitation.2015.07.038>. Accessed 08 Jun 20.

[https://www.ahajournals.org/toc/circ/132/18\\_suppl\\_2](https://www.ahajournals.org/toc/circ/132/18_suppl_2). Accessed 08 Jun 20.

# Appendix B: Comparing and Selecting Portable Emergency and Transport Ventilators

## B.1 Introduction

There has been a rapid advance in the design, production and use of portable automatic ventilators over the past 30 years. The result is that there is now a bewildering array of devices on the market covering a wide range of complexity and price. Faced with this situation selecting a suitable portable ventilator from the range available for a specific medical or paramedical service is not easy and there are many factors that should go into making a choice, either for an individual or for a large scale procurement. Examples of various resuscitation and emergency/transport ventilators were presented in Chap. 7. The published technical specifications of these will be considered in Appendices C and D. This appendix covers the essential points that should be borne in mind before selecting and using a portable ventilator.

## B.2 Classes of Available Portable Mechanical Ventilators and Examples

Chapters 7–12 have discussed a number of ventilators that may be used in emergency and transport ventilation as well as in the management of mass ventilation. In terms of classification these may be conveniently be divided into resuscitation, emergency and transport ventilators. These classes are to some extent arbitrary and the use of ventilator will depend upon many factors such as availability, setting and the training and experience of the operators. Also, as has been seen in earlier chapters of this book ventilators may have uses outside of the intended use, again

depending on the setting and availability. Thus ventilators classed as ‘resuscitation ventilators’ may be used effectively in short-term transport and in mass emergency ventilation when no other devices are available.

### ***B.2.1 Resuscitation Ventilators (Single Tidal Volume/ Frequency Control, Pneumatically Powered, May Be Either Volume or Pressure Generators)***

This type of basic ventilator was discussed in Sect. 7.2.1. Essentially these devices are hand-held and are designed to provide immediate life-saving ventilation using only a minimum of controls by responders with basic training in life support. They provide more reliable ventilation than is possible with the bag-valve-mask.

### ***B.2.2 Emergency/Immediate Transport Ventilators (Separate Tidal Volume and Frequency Controls, Time Cycled, Volume Preset, Pneumatically Powered)***

This type of ventilator was discussed in Sect. 7.2.2 and is suitable for use by non-specialists in both emergency and immediate transport situations (for example, at the scene of an emergency and during primary transport to hospital. They are also used inside the hospital in the ER and for transport between the ICU and other facilities such as imaging. (Note that MRI compatibility should be checked against the manufacturers’ specifications.

## **B.3 Choosing a Portable Ventilator**

Faced with the wide range of ventilators currently available on the market, choosing which device will be the most suitable for any one emergency or hospital service may be a daunting task. This section considers some of the factors which go into choosing a ventilator and how best to approach the task.

### ***B.3.1 Factors Determining the Choice of Ventilator. These Include the Following:***

- Setting and tasking—where and how the ventilator will be used
- Planned requirements—how many units are required? Is there to be a stockpile for mass ventilation requirements?
- Who will use the ventilator?

- Training and familiarity with the device in non-emergency situations
- Supply—availability of suitable agents for the manufacturers and whether the manufacturer can supply large numbers of ventilators in the case of stockpiling. This proved to be a problem in some countries during the COVID 19 pandemic.
- Servicing—can the supplier provide regular servicing for the ventilator. This may be difficult in remote locations.
- Costs—these include both the initial outlay and the servicing charges. Apart from cost of the ventilator there may be added continuing costs such as covering the supply of disposable equipment such as filters and patient circuits. Check also that the disposables are available for resupply during mass ventilation.

### ***B.3.2 Who Should Select the Ventilator?***

The selection of the most suitable ventilator should ideally rest with the persons who will be using it. For those unfamiliar with portable ventilators specialist help may be available within the hospital from anaesthetists and intensivists who use ventilators on a regular basis. Such expertise will be helpful in assessing the published technical data.

Allowing the purchase of ventilators to be only in the hands of accounting personnel is not an ideal situation. Purchase of equipment, particularly for stockpiling ventilators for mass ventilation is usually done by tender. There is always the temptation by those in charge of funding to go for the cheapest tender without considering the quality of ventilation that will be provided. Be aware that not all portable ventilators are the same and there is considerable variation in function, particularly in pressure generators (see Chap. 7). These ventilators should not be used in emergency. In certain conditions of increased airway resistance and decreased lung compliance they may deliver inadequate tidal volumes or even may not work at all, jamming during the cycling.

If it is not possible for the potential user to have the final voice in deciding which ventilation should be bought they should at least be part of committee or other body who makes the purchasing decision.

### ***B.3.3 Gathering Information Before Buying***

Manufacturers will published a certain amount of information about a product in their promotional literature. This may not always cover the function and limitations of the device and it is important to gather as much additional information as possible. The operator's handbook will usually contain much more information about the design and function of the device and its technical specification and also providing careful instructions about how the device should be used. Try to see a handbook for the device being considered before buying.

Other useful information can be obtained from manufacturers' sales teams. These will visit potential customers in emergency and hospital services on a regular basis and can also be helpful during trade exhibitions which usually accompany medical congresses. It is always worth visiting such exhibitions to be able to compare the ventilators on display. The following points are important when dealing with sales teams:

- Beware of inflated claims about the ventilator
- Do not be sold something you do not require!
- Always try to see a ventilator working (for example with a test lung) and try the controls before considering purchase further (many manufacturers now have video demonstrations available on their web sites which can provide useful information).
- Check that the device fully conforms with the regulations of the country where it is to be used. Medical devices must be tested and approved by regulatory bodies such as the Federal Drug Administration in the US. Usually manufacturers will not try to sell a device in a certain country unless full approval has been obtained. This can often be a lengthy and costly business.

There are a number of papers which compare various portable ventilators currently available on the market and these are listed in the suggestions for further reading at the end of this appendix.

### ***B.3.4 Testing and Commissioning a Ventilator***

If purchasing multiple ventilators (for example in re-equipping an ambulance service or for mass ventilation always try to buy a single ventilator first and submit it to internal testing by your own service. This is usually done against a calibrated test lung to compare parameters such as delivered tidal volume and oxygen concentration during airmix with the manufacturer's published data.

Testing a ventilator in the controlled setting of the anaesthetic room is very valuable. Patients who have been anaesthetized and are stabilized on operating theatre ventilators and monitoring equipment can be safely switched to the ventilator being trialled knowing that full back up ventilation and monitoring is available should any problems be encountered. The operator should be fully familiar with the controls and function of the ventilator to be tested by using it on a manikin or test lung before working in a clinical environment.

One problem that may arise in certain countries however is that portable ventilation equipment designed for use in emergency may not be approved for use in hospital settings. This has been a problem in the past with training on field anaesthetic systems. Always check the local protocols before using a new piece of equipment therefore.

### ***B.3.5 Training and Use***

Once a ventilator has been purchased a complete training programme for those who are to use it should be started. Manufacturers will usually have their own training staff who can train those who are to be involved in the normal medical training) on a ‘train the trainers’ basis). Potential ventilators users who have only used bag-valve devices in the past will possibly require extra reassurance about the function of the mechanical ventilator with which they may not be at all familiar. This underlies the importance of buying a ventilator that is suitable for the purpose in hand without having too many unnecessary controls.

It is important that portable ventilators should be used as regularly as possible. Their advantages over bag valve ventilation, both in ease of use and the quality of ventilation provided have been discussed earlier in this book but it is important that these advantages should be recognized.

If ventilators are to be stockpiled for use in mass ventilation they should be individually checked and commissioned before stockpiling and, importantly checked on a regular basis in accordance with the manufacturer’s servicing recommendations. This is important whether or not the ventilators have been used.

#### **Suggestions for Further Reading**

- Branson R. Patient needs should drive ventilator selection for stockpiling; ‘handy’ ventilators may not ‘lend a hand’. *Respir Care*. 2011;56(6):879
- Chipman DW, Caramenz MP, Miyoshi E, et al. Performance comparison of 15 transport ventilators. *Respir Care*. 2007;52(6):740–51.
- Johannigman JA, Branson RD. Ventilatory support in the field. *Respir Care Clin N Am* 1996;(Sept):353.
- L’Her E, Roy A. Bench tests of simple, handy ventilators for pandemics: performance, autonomy and ergonomics. *Respir Care*. 2011;56(6):751–67.
- L’Her E, Roy A, Marjanovic N. Bench – test comparison of 26 emergency and transport ventilators. *Crit Care*. 2014;18:506–19.
- Tempier F, Miroux P, Dolveck F, et al. Evaluation of the ventilator – user interface of 2 new advanced compact transport ventilators. *Respir Care*. 2007;52(12):1709–10.

## Appendix C: Technical Specifications of Selected Resuscitation Ventilators

	Pneupac VR1	MicroVENT Professional Adult and child resuscitator	Carevent BLS/ALS resuscitator	Oxylator EMX
Manufacturer	Smiths Medical International, UK	Meditech, UK	O – two systems, Canada	CPR Medical Devices Inc, Canada
Manufacturers' intended use statements	The Pneupac VR1 is a hand—held portable ventilator intended for use in emergency or transport situations only where the patient is constantly monitored by the user. It offers both automatic and manual release ventilation. The VR1 is intended for emergency resuscitation by medical personnel, paramedics and ambulance technicians inside and outside hospital.	The MicroVENT is a pneumatically powered, time/ volume cycled resuscitator with manual triggering for use in conjunction with external cardiac massage, automatic cycling being provided for longer term ventilatory support or patient transport.	The Carevent BLS/ALS resuscitators are hand held devices designed for emergency resuscitation and inter-departmental transport in the hospital environment where the potential patient use is with children and adults.	The Oxylator EMX is a pressure limited, flow triggered ventilation device designed to replace the BVM during cardiopulmonary resuscitation and transport. ( <i>The manufacturers state that the device is not a demand valve or ventilator but is a patient responsive positive pressure device</i> )
Target population	Above 10 kg (22 lb)	Above 20 kg (44 lb)	Above 20 kg (44 lb)	Above 10 kg (22 lb)

	Pneupac VR1	MicroVENT Professional Adult and child resuscitator	Carevent BLS/ALS resuscitator	Oxylator EMX
Pneumatic power source	Dry, oil free, filtered oxygen	Oxygen or air	Dry, oil free, filtered gas: Oxygen or air	Oxygen
Input gas pressure	276–1034 kPa (40–150 psi)	45–70 psi (3–5 bar)	345–1034 kPa	3.5 bar (50 psi)
Patient interface	360° swivelling, reusable, removable patient valve with a flutter valve at the expiratory port. A secondary circuit can be attached to allow ventilation via an ETT	360° swivelling, reusable, removable patient valve with a flutter valve at the expiratory port	360° swivelling, removable patient valve	Mask/ETT connection
Operating modes	Automatic Manual (selected by mode selector)	Automatic Manual (selected by mode selector switch)	Automatic Manual Off (selected by seven position selector lever)	Manually active and automatic
Display and user controls	Tidal volume/frequency control Mode selector dial Manual push button and omni-directional lever	Tidal volume/frequency control Mode selector switch Manual trigger	Tidal volume/frequency control and mode setting selector switch as above with viewing window Manual button	Rotary minute volume control
Method of operation	Time-cycled, volume-preset flow generator, pressure limited	Time/volume cycled resuscitator with manual triggering, pressure limited	Pneumatically powered, pressure limited time/volume cycled ventilatory resuscitators	Pressure cycled between 25 and 50 cm H <sub>2</sub> O with a constant flow of 40 l/min
Relationship between tidal volume and frequency control	Interdependent	Independent	Interdependent	n/a



	Pneupac VR1	MicroVENT Professional Adult and child resuscitator	Carevent BLS/ALS resuscitator	Oxylator EMX
Tidal/minute volume	A continuous range of tidal volume/frequency with calibration points marked at; 150 ml 300 ml 450 ml 600 ml (detent) 750 ml 900 ml 1050 ml	Continuous tidal volume range from 300 to 1200 ml	Tidal volume range 150–600 ml (GOS) 200–1100 (Standard)	Minute volume range 10–12 l/min in automatic mode
Frequency	A continuous range of tidal volume/frequency with calibration points marked at; 25 bpm 20 bpm 15 bpm 12 bpm (detent) 11 bpm 10 bpm 10 bpm	Continuous range from 12–24 bpm (detent 12)	12–20 bpm (detent @ 12 bpm)	Ventilation frequency auto adjusting to lung capacity
I:E ratio	1:2	1:2	1:2	1:1 to 1:2
Flow rate	Output flow rate is determined by the position of the tidal volume/frequency slider and varies between approximately 11–32 l/min	21.6–43.2 l/min in automatic mode (32 l/min at detent)	12–40 l/min. in automatic mode and fixed in manual mode (Standard)	Constant flow at 40 l/min
Oxygen concentration	100% oxygen	100% oxygen	100% oxygen	100% oxygen
Air mix	50% oxygen			Adjustable 0–50% oxygen via built-inhalator

	Pneupac VR1	MicroVENT Professional Adult and child resuscitator	Carevent BLS/ALS resuscitator	Oxylator EMX
Demand function	Demand flow initiated by breathing effort $<-2$ cm H <sub>2</sub> O in compliance with ASTM F920-93 Demand function also includes ventilator inhibit feature	Respiratory assist whereby patient inhalation triggers restart of inspiratory cycle with approx. $-5$ cm H <sub>2</sub> O	Demand flow initiated by breathing effort $<-2$ cm H <sub>2</sub> O Demand function also includes ventilator inhibit feature	n/a
Method of operation in manual mode	In manual (MAN) mode, the user may initiate single breaths up to the selected tidal volume by pressing down the VENT push button or moving the omni-directional lever in any direction. If the user delivers the whole set tidal volume, the ventilator will 'lock out' until the appropriate E time has elapsed, whereas if a breath is delivered that does not exceed the set tidal volume, further breaths may be given until the whole set tidal volume has been used.	Operation of manual control provides a continuous flow of gas at 40 l/min with patient pressure limited by relief pressure valve	Operation of manual control provides a continuous flow of gas at 40 l/min with patient pressure limited by relief pressure valve	Operation of manual control provides a constant flow of 40 l/min
Relationship to tidal volume and frequency control setting when in manual mode	Maximum tidal volume delivered is limited by position of rotary control thus protecting the patient from stacked breaths and over-inflation	None	None	None

	Pneupac VR1	MicroVENT Professional Adult and child resuscitator	Carevent BLS/ALS resuscitator	Oxylator EMX
Flow rate in manual mode	Output flow rate is determined by the position of the tidal volume/frequency slider and varies between approximately 11–32 l/min	40 l/min	40 l/min	40 l/min
Spontaneous breathing under power failure	Full spontaneous breathing under power failure with flutter valve permanently fitted	On advanced models, the anti-inhalation valve is fitted so spontaneous breathing under power failure is not possible	Spontaneous breathing under power failure available by omission of anti-air inhalation valve (but entrains surrounding atmosphere)	Not specified
Airway Pressure Limiting System	Pressure relief valve with audible pneumatic alarm set to 40 cm H <sub>2</sub> O (60 cm H <sub>2</sub> O optional)	Pressure relief valve with audible pneumatic alarm set to 45 cm H <sub>2</sub> O (60 cm H <sub>2</sub> O optional)	60 cm H <sub>2</sub> O	25–50 cm H <sub>2</sub> O
MRI compatibility	MR Compatible to 3 Tesla and field gradient of 430 G/cm.	No	No	No
PEEP capability	PEEP available with external PEEP adapter when using a patient circuit (secondary configuration)		PEEP available with external PEEP adapter	2–4 cm H <sub>2</sub> O
Height	95 mm (including patient valve)	90 mm	106 mm	108 mm
Width	100 mm	55 mm	64 mm	57 mm
Depth	170 mm	120 mm	150 mm	
Weight	400 g	272 g	400 g	250 g
Gas consumption	Delivered tidal volume plus approx. 40 ml/breath	Quoted as insignificant	Not specified	Quoted as zero

	Pneupac VR1	MicroVENT Professional Adult and child resuscitator	Carevent BLS/ALS resuscitator	Oxylator EMX
Duration of gas supply	At 12 bpm and 0.6 l V <sub>T</sub> approx. 40 min with ‘D’ size cylinder containing 415 l of oxygen at STP (15 °C and sea level)	At mid-setting 34 min with ‘D’ size cylinder containing 370 l	At 20 bpm and 0.2 l V <sub>T</sub> approx. 100 min with ‘D’ size cylinder containing 415 l of oxygen At 12 bpm and 1.1 l V <sub>T</sub> approx. 30 min with ‘D’ size cylinder containing 415 l of oxygen	Min time of oxygen supply = cylinder volume divided by 12 l/min

Source: Manufacturers’ published literature, June 2020

# Appendix D: Technical Specifications for Selected Emergency/Transport Ventilators

Ventilator name	Allied MCV 200	Carevent MRI	Medumat Transport	Osiris 3	Oxylog VE300	pNeuton	Pneupac Parapac Plus 310
Manufacturer	Allied Health Care Products Inc	O – Two Medical Technologies Inc	Weinmann Medical Technology	Taema – Air Liquide	Dräger Medical AG	Airon Corporation	Smiths Medical International
Country	USA	Canada	Germany	France	Germany	USA	United Kingdom
Ventilator type	Pneumatic/ Electronic time cycled constant flow	Pneumatic	Pneumatic/ electronic	Electronic	Time- controlled, constant volume, pressure- monitored	Pneumatic time-cycled volume preset	Pneumatic time cycled volume preset flow generator
Ventilation modes	Assist Control Ventilation Manual breath button	Spontaneous Manual A/C CPAP	Controlled Ventilation Mode Assisted Ventilation mode Intermittent Positive Pressure Ventilation CPAP	A/C CMV Spont Manual	IPPV SIPPV A/C, PS Manual Spontaneous CPAP	CMV IMV CPAP Spontaneous	CMV/demand Manual release Free flow oxygen CPAP Neonatal Ventilation circuit
PEEP	n/a	PEEP—0– 20 cm H <sub>2</sub> O	Yes	0–15 cm H <sub>2</sub> O	0–20 cm H <sub>2</sub> O	0–20 cm H <sub>2</sub> O	Internal adjustable 0–20 cm
Gas supply sources	Central Gas Supply System Medical Gas Cylinder	Central Gas Supply System Medical Gas Cylinder	Central Gas Supply System Medical Gas Cylinder	Central Gas Supply System Medical Gas Cylinder	Central Gas Supply System Medical Gas Cylinder	Central Gas Supply System Medical Gas Cylinder	Central Gas Supply System Medical Gas Cylinder

Ventilator name	Allied MCV 200	Carevent MRI	Medumat Transport	Osiris 3	Oxylog VE300	pNeuton	Pneupac Parapac Plus 310
Supply gases	Medical Oxygen Medical Air	Medical Oxygen Medical Air	Medical Oxygen Medical Air	Medical Oxygen Medical Air	Medical Oxygen Medical Air	Medical Oxygen Medical Air	Medical Oxygen Medical Air
Supply pressure	280–600 kPa(2.8–6.0 bar)	45–70 psi	2.7–6 bar	2.8–6 bar	2.9–7.0 bar/40–100 psi	55 ± 15 psi (3.8 bar)	280–600 kPa at 65 l/min
Oxygen consumption	7.1 l/min	10.0 l/min 5.2 l/min (airmix)	<i>Not specified</i>	11.4 l/min 5.3 l/min(airmix)	Stated as ‘low’ due to Duroflow™ Technology	Table of consumption in user’s manual. 45 min on CPAP with ta D-sized cylinder	With a MV of 10 l/min 60 min duration from a 680 l E cylinder delivering 100% oxygen. 160–180 min on airmix (50% oxygen)
Electrical supply	115 V/240 V 50/60 Hz 2 AMPS AC	n/a	3.6 V Lithium Battery Button cell CR2430 for auxiliary power	100–230 V 50–60 Hz 8–30 V DC	19 V battery with 230 V DC converter	n/a	3.6v Lithium battery for monitoring system (special low magnetic AA battery for MRI and general use)
Battery life (hours)	30 h on Oxygen 7 h on Air	n/a	2 years under normal operating conditions	6–14 h	Max 8 h NiCd	n/a	3000 h under normal operating conditions
Battery charge time	5 h if off 10 h if in use		n/a	3 h	5 h (battery exchangeable)	n/a	n/a
Dimensions	368.3 mm × 261.1 mm × 88.9 mm	100 mm × 240 mm × 194 mm	110 mm × 190 mm × 90 mm	210 mm × 250 mm × 270 mm	399 mm × 153 mm × 160 mm	100 mm × 200 mm × 150 mm	93 mm × 240 mm × 165 mm
Weight	7.7kg		1.1 kg	5 kg	3.6 kg	2.7 kg	2.4 kg
Air mix range	65–100%	60–100%	55–5% at 10 mbar	50–100%	100% O <sub>2</sub> + airmix (actual value delivered depends on inspiratory flow and mean airway pressure)	65–100%	50–100%

Ventilator name	Allied MCV 200	Carevent MRI	Medumat Transport	Osiris 3	Oxylog VE300	pNeuton	Pneupac Parapac Plus 310
Vt range	200–1200 ml	Minute volume 2–14 l	50–2000 ml	100–2000 ml	100–2000 ml	360–1500 ml	150–1500 ml (colour-coded controls to match frequency and tidal volume settings for maximum accuracy)
Frequency range			0–60/min		5–50 bpm		8–40 bpm (détente at 12 bpm)
I:E ratio	Possible to set 1 s or 2 s inspiration time	I:2	59:1–1:59	1:3–1:1	1:4–4:1	Inspiration time 0.6–2.5 s Expiration time 0.6–30 s	1:1.4–1:2.5
Display	LCD	n/a	LCD colour display	LCD screen	LCD colour display	n/a	n/a
User adjustable alarms	High and low pressure alarms Airway Pressure Relief	Airway over pressure alarm	3–60 mbar	Upper and lower airway pressure limits F max Min expired Vt	Apnoea, High and Low inflation pressure, respiratory rate	High pressure alarm	High and low pressure alarms (visual and audible)
Additional alarms	Low source gas alarm	Circuit disconnect Alarm—visual/audible	In assist control ventilation returns if patient fails to trigger over two cycles 5 audible alarms 5 LED	Disconnect alarm Power failure alarm Battery fully charged indicator Loss of PEEP	Et CO <sub>2</sub> (option), Disconnection	Low pressure alarm Anti suffocation system	Low gas source indicator and alarm Audible alarms silenced for 60 s—visual indicator
Alarm indicators	1-RED LED Audible alarms for high/low pressure Low Battery LED Indicator	Visual coloured light Audible sound	Coloured display	4 LEDS	Audible low a/w pressure alarm	<i>Not specified</i>	LED indicators for high and low pressure, cycling and audible alarms silenced
MRI compatibility	No	MRI 3 Tesla	No	No	No	MRI 1.5 Tesla	Yes—to 3 Tesla

Source: Manufacturers' published literature, June 2020

## Appendix E: Ventilation Modes Used in the Intensive Care Unit

This appendix gives a synopsis of the modes of ventilation in the ICU. At present non-specialist providers of IPPV are not likely to be asked to use these. However as emergency and transport ventilators become more developed in the future with the need to provide adaptive ventilation, a basic understanding of what is currently available in the ICU will be increasingly useful. Modern complex ventilation modes are best described dynamically and most manufacturers now include video presentations of ICU ventilators on their web sites. The interested reader is advised to consult these for more information.

### E.1 Volume Assist Control (or Assisted Controlled Mandatory Ventilation (ACMV))

This is a widely-used ventilation mode in the ICU where the ventilator interacts with the patient's own respiratory efforts through a triggering system. This is the equivalent of the CMV-demand mode found on many pneumatic emergency ventilators.

In ACMV a preset  $V_t$  is delivered at a set rate thus ensuring a set and adequate minute volume.

Advantages of ACMV

1. Adequate ventilation is potentially assured provided that the backup rate has been set high enough
2. Allows the patient to rest by relieving him of most of the work of ventilation
3. Does not preclude spontaneous breathing by the patient.
4. Each breath is delivered in synchrony with the patients own respiratory effort



### Disadvantages of ACMV

1. The breathing schedule is relatively rigid and may lead to alkalosis if the rate is set too high
2. If the lung compliance is low the airway pressure will rise. This is countered by a high pressure release valve (as on the Parapac Plus)
3. The trigger settings by the operator are important otherwise the work of breathing by the patient can be too high.
4. Using this mode in the ICU, sedation is often required
5. Atrophy (wasting) of the respiratory muscles can occur if the mode is used over extended periods of time.

## **E.2 Intermittent Mandatory Ventilation (IMV) and Synchronised Intermittent Mandatory Ventilation (SIMV)**

IMV and SIMV were discussed in Chap. 6. These modes have been used along with ACMV in the past and a comparison of the two approaches is useful.

1. SIMV was originally used to wean patients off ventilation in the days before complex pressure targeted interactive modes. It is less used in the ICU now but as noted earlier is a feature on some emergency and transport ventilators.
2. In SIMV mandatory breaths are set by the physician, irrespective of the patients own demands.
3. In between the mandatory breaths the patient may take spontaneous breaths depending on the respiratory effort available. If the backup rate has not been set high enough hypoventilation and hypoxia may occur due to too low a minute volume.
4. With the original CMV mode, clashing of mandatory and spontaneous breaths could occur. To overcome this synchronised intermittent mandatory ventilation was developed.

### Important differences between SIMV and ACMV

1. In SIMV the patient receives the full complement of the preset mandatory ventilation, irrespective of how many spontaneous breaths he takes.
2. In ACMV the patient determines his own respiratory rate provided that the patient is breathing at a rate above the set backup rate.
3. In ACMV each breath, whether patient triggered or a ventilator back-up breath is delivered at a preset tidal volume

### Key points about SIMV vs. ACMV

1. SIMV encourages the patient to use his respiratory muscles which is a great advantage in weaning the patient off a ventilator

2. ACMV is very different in this respect. Here, the patient performs very little of the work of breathing. Once triggered, the ventilator is responsible for the entire work of breathing allowing resting of exhausted respiratory muscles. ACMV is therefore used in the ICU where a greater degree of respiratory support is often needed.

### **E.3 Pressure Support Ventilation**

1. In pressure support ventilation (PSV) the ventilator augments the respiratory efforts of the patient. Exhalation is passive
2. The delivered tidal volumes are a function of the operator—preset level of pressure support
3. In PSV, used in the ICU the patient controls the depth, length and flow profile of each breath.

The controlling factors in PSV—what is happening?

With the onset of inspiration, the airway pressure rapidly rises and this level of pressure is maintained for most of the inspiratory period. When the airflow slows down towards the end of inspiration, the ventilator cycles to expiration and passive expiration follows. The threshold for the change between inspiration and expiration is dependent on the gas flow to the patient. In most PSV ventilators this is 25% of the peak flow.

As with other modes of support ventilation there is a back up cycling. This is time cycled. This back up is necessary in case there is a leak in the ventilator circuit and flow cycling does not occur. In this situation the inspiration is terminated after a set time of 3–5s.

It must be noted that PSV will only work if the patient is capable of a respiratory effort sufficient to activate the trigger. Setting the triggering is a key step in ICU ventilation unlike emergency ventilation.

#### ***E.3.1 Advantages of Pressure Support Ventilation***

1. The patient can breathe at the respiratory rate he chooses.
2. The patient controls the inspiratory time and flow of each breath. This is an important distinction from pressure control ventilation
3. Because the patient is in control PSV is largely comfortable and well-tolerated. There is perfect synchronisation (by definition) with the ventilator.
4. As its name suggests PSV essentially supports the inspiratory effort of the patient. Over a period of time, with enhancement of tidal volumes as the lungs improve the respiratory rate falls off. This is a good clinical sign of more comfortable breathing

5. A low level of pressure support (5–10 cm H<sub>2</sub>O) usually overcomes the extra airway resistance caused by the use of an endotracheal tube.

### **E.3.2 Disadvantages of Pressure Support Ventilation**

1. Only pressure in the airway is assured and not tidal volume. With a falling lung compliance and increased airway resistance this may be a major problem. If the patient still has respiratory drive there can be compensation by raising the respiratory rate to counteract falling tidal volumes
  2. PSV is poorly-supported in patients with active bronchospasm
  3. Patients with chronic obstructive airway disease can easily become desynchronised with the ventilator as follows:
    - Narrowed airways result in slower lung filling during inspiration
    - Late into inspiration the flow rate remains above the cycling threshold
    - The ventilator fails to cycle to expiration even though the patient's neurological inspiratory time has been completed
    - The patient attempts to exhale while the ventilator is still delivering a breath
- Patient–ventilator desynchronisation occurs

## **E.4 Other Modes of Ventilation Used in the ICU**

With the development of the microprocessor-controlled ventilator manufacturers have introduced other complex modes of ventilation some of which are listed here. There is a bewildering range! Essentially these are variations on the two basic systems of volume and pressure-targeted ventilation which are described above. Often, the ventilator manufacturers have patented variation in the standard modes for commercial purposes.

The overall trend with the development of complex modes of ventilation is to make ventilation as interactive with the patient as possible to improve patient tolerance of artificial ventilation and the efficiency of the ventilation itself.

It is essential for the function of all the interactive ventilation modes that the patient still has some respiratory effort i.e. is not in respiratory failure as in cases requiring emergency ventilation.

### ***E.4.1 Airway Pressure Release Ventilation (APRV)***

Also called:

- CPAP with release
- Intermittent CPAP
- Variable Positive Airway Pressure (VPAP)

APRV involves the periodic release of pressure while breathing in the CPAP mode. The release may be time-cycled or occur after a set number of breaths. This form has been termed Intermittent Mandatory Airway Pressure Release Ventilation (IMPRV).

The indications for this mode are similar for pressure controlled ventilation. The mode is said to improve lung recruitment in spontaneously breathing patients and is also said to produce better synchrony with the ventilator.

### ***E.4.2 Proportional Assist Ventilation (PAV)***

In PAV pressure, flow and volume are dictated by the patients breathing efforts analysed by microprocessor. In addition the clinician determines how much work the ventilator must do the amplify the patient's respiratory efforts in relation to the prevailing lung mechanics. As the name suggests the ventilator can be set by provide a set proportion of the work of breathing. A typical initial setting is 80%.

### ***E.4.3 Dual Breath Control***

Many modern ICU ventilators now incorporate complex computer algorithms capable of controlling simultaneously two variables. There are two forms:

- Interbreath control
- Intrabreath control

#### **E.4.3.1 Intrabreath Control**

Also known as: Dual control within a breath (DCWB)

In this mode, the clinician sets the minimum required tidal volume and the backup breath flow and rate. The breaths from the ventilator can be patient or machine triggered. The configuration of the breath depends on the patient's own respiratory efforts. When the patient effort is satisfactory the conformation of the DCWB waveform becomes similar to a standard pressure support breath i.e. the flow is decelerating and is terminated by flow cycling. If the patient's breathing is unsatisfactory (inadequate Vt) the machine delivers controlled breaths that are flow - targeted and volume cycled.

#### **E.4.3.2 Interbreath Control**

In this mode, flow and inspiratory time are automatically adjusted to deliver a targeted Vt with the lowest peak inspiratory pressure possible.

#### ***E.4.4 Pressure Regulated Volume Control (PRVC)***

This mode is essentially a variant of the standard pressure control (PC) mode.

In this mode the ventilator delivers a test breath which, on the basis of the delivered tidal volume calculates the current lung compliance. Each breath is then delivered after calculating the compliance for the preceding breath. The flow and inspiratory time are automatically adjusted to deliver the targeted  $V_t$  with the lowest inspiratory pressure possible.

#### ***E.4.5 Automode***

This is a mode where the patient can switch between a support mode and a control mode. An absence of triggering (i.e. 12 s of apnoea in adults) causes the ventilator to move over to control mode.

#### ***E.4.6 Volume Support***

This mode essentially uses pressure support breaths but with microprocessor-controlled modification. The pressure support for each breath is calculated from each preceding breath and the inspiratory pressure is regulated so that a set  $V_t$  is delivered in other words, as elsewhere  $V_t$  is guaranteed.

This mode is an example of volume targeted ventilation within the pressure support mode and provides an example of how microprocessor - controlled ventilators in the ICU are crossing the standard lines of demarcation between volume and pressure targeted ventilation.

#### ***E.4.7 Adaptive Support Ventilation***

This mode represents the most sophisticated form of ventilation available today. It is a totally interactive mode which combines many of the features of the modes described previously. Input from the clinician is only to key in the patient's weight and whether the patient is adult or child. The ventilator then does the rest, delivering breath by breath modified ventilation according to changes in the patient's lungs. It takes into account system compliance and resistance, auto PEEP. It selects the appropriate parameters (frequency, upper airway pressure limit, I:E ratio, inspiratory time for both mandatory and spontaneous breaths. Machine-delivered backup if there is no effort from the patient delivers the basic machine triggered, time cycled mandatory breaths necessary to provide an adequate  $V_t$  (See Chap. 11, Sect. 11.7.2).

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